

A *Daubert* Analysis of Abusive Head Trauma/Shaken Baby Syndrome—Part II: AN EXAMINATION OF THE DIFFERENTIAL DIAGNOSIS¹

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Introduction

For reasons inexplicable to many physicians, and unbeknownst to many others, the diagnosis of Abusive Head Trauma/Shaken Baby Syndrome² (AHT/SBS) remains a lightning rod for controversy. Public media articles continue to be published.³ Legal articles continue to be written.⁴ And, judicial commentary on the science continues to occur.⁵ The most recent example of judicial commentary upon the topic, and probably the most prominent, is the dissenting opinion of the honorable Justices Ginsburg, Sotomayor, and Breyer in *Cavazos v. Smith*.⁶ In that opinion, the dissenting justices cited seven medical articles that ostensibly supported their opinions that: 1) “there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to SBS”; 2) “that ‘the commonly held opinion that the finding of [subdural hemorrhage] and [retinal hemorrhage] in an infant was strong evidence of SBS was unsustainable’”; and, 3) that

¹ **THIS ARTICLE IS NOT FINALIZED.** The following footnotes have not been substantiated and may be incomplete or incorrect.

² The authors’ use of the terminology “Abusive Head Trauma/Shaken Baby Syndrome” is not to imply that the terms are interchangeable. It is simply to recognize that there is a commonly recognized subset of Abusive Head Trauma—Shaken Baby Syndrome—that is the primary subject of controversy, and that pediatricians have transitioned to a more encompassing term—Abusive Head Trauma. See, e.g., Emily Bazelon, *Shaken Baby Syndrome Faces New Questions in Court*, N.Y. TIMES, Feb. 2, 2011, available at http://www.nytimes.com/2011/02/06/magazine/06baby-t.html?_r=1.

³ *Id.*; Deborah Tuerkheimer, *Anatomy of a Misdiagnosis*, N.Y. TIMES, Sept. 20, 2010, available at www.nytimes.com/2010/09/21/opinion/21tuerkheimer.html?ref=opinion.

⁴ See generally Keith A. Findley et al., *Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right*, 12 Hous. J. HEALTH L. & POL’Y 209 (2012); Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. U. L. REV. 1 (2009).

⁵ See, e.g., *State of Wisconsin v. Edmunds*, 746 N.W.2d 590 (Wis. Ct. App. 2008) (finding that a “significant and legitimate debate in the medical community has developed in the past ten years”); *Hamilton v. Commonwealth of Kentucky*, 293 S.W.3d 413 (Ky. Ct. App. 2009) (holding error to permit testimony on shaken baby syndrome without first conducting a *Daubert* hearing since no Kentucky case had specifically determined it was a “reliable” theory).

⁶ *Cavazos v. Smith*, 132 S.Ct. 2, 10 (2011).

“doubt has increased in the medical community ‘over whether infants can be fatally injured through shaking alone.’”⁷

Setting aside the multiple concerns regarding the selection criteria for the articles,⁸ the irony in the citation of these articles is that the articles cited by the dissenting justices are actually so methodologically flawed, scientifically inaccurate, and of the lowest level of evidence-based medical literature, that they would be reasonable examples of articles that are “not even good enough to be wrong.”⁹ So how do the most learned jurists in the land get the science so wrong?¹⁰ And what hope is there then for the lone “gatekeeper”?

In Part I of this discussion, one of the authors, Dr. Narang, presented a relatively comprehensive analysis of the current science surrounding AHT/SBS, and more specifically, surrounding two of the most common injuries found in AHT/SBS—subdural hemorrhages (SDHs) and retinal hemorrhages (RHs).¹¹ Dr. Narang asserted that the diagnosis of AHT is supported by “at least 700 peer-reviewed, clinical medical articles comprising thousands of pages of medical literature, published by over one thousand different medical authors, from at least twenty-eight different countries.”¹² He described, in painful detail, multiple scientific studies, from various medical disciplines, that demonstrated a significant statistical association of SDHs with AHT¹³ (over accidents and other medical causes) and that demonstrated a highly significant statistical association of severe RHs with AHT¹⁴ (over accidents and other medical causes). Despite the reported “controversy” on the topic, Dr. Narang cited at least fifteen international and national professional medical societies that have publicly acknowledged the validity of AHT either through formal practice statements or through educational materials provided to their members or the public.¹⁵ In conclusion, Dr. Narang examined that scientific literature with *Daubert* scrutiny and argued that such literature was scientifically valid, and

⁷ *Id.*

⁸ J. Moreno & B. Holmgren, *Dissent into Confusion: The Supreme Court, Pseudoscience, and the False Shaken Baby Syndrome Controversy*, UTAH L. REV. (forthcoming 2013).

⁹ Stephen Breyer, *Introduction*, Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, 1, 4 . 2d ed. 2000), available at [www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/\\$file/sciman00.pdf](http://www.fjc.gov/public/pdf.nsf/lookup/sciman00.pdf/$file/sciman00.pdf).

¹⁰ Moreno & Holmgren, *supra* note 7. (providing a detailed analysis of the rationales and shortcomings of the dissenting justices’ opinion in *Cavazos v. Smith*).

¹¹ See Sandeep Narang, *A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome*, 11 HOUS. J. HEALTH L. & POL’Y 505, 505 (2012).

¹² *Id.* at 578.

¹³ *Id.* at 541-48.

¹⁴ *Id.* at 548-58.

¹⁵ *Id.* at 574-76.

consequently, did provide the clinician with sound scientific basis for arriving at diagnosis of AHT.¹⁶ However, as Dr. Narang alluded to in his initial article, that was only part of the analysis.

Part II of this discussion swings the microscope in the opposite direction. The differential diagnosis of AHT (and its two most common injuries, SDHs and RHs) includes many things—accidental trauma, birth trauma, bleeding disorders, malignancy, and metabolic/genetic syndromes, to name a few.¹⁷ Although a detailed scientific analysis¹⁸ of the entire differential diagnosis is beyond the purpose and scope of this article, this paper will examine the best, current evidence-based data for two of the most common items on the differential diagnosis, accidental injury and bleeding disorders and then examine the evidentiary basis for two of the most debated topics in AHT, biomechanics and hypoxia/ischemia. In being provided with the “rest of the story,” the reader will, hopefully, be able to see the relative strengths and weaknesses of the scientific data underpinning SDHs, RHs, and the differential diagnosis. The reader will thereafter be able to discern for himself/herself whether the scientific data afford the clinician reasonable grounds for arriving at the diagnosis of AHT. More importantly, the reader will be provided with a reasonable glimpse of the entire analysis—the *methodology*—a clinician undertakes in arriving at the AHT diagnosis and be able to conclude for himself/herself whether that methodology is reasonable or, simply, “junk science.”

In the first subsection of this paper, we briefly review the concept of “evidence-based medicine,” discuss its proper role in present-day clinical medicine, and proffer an acceptable ranking scale for evidence-based medical literature,¹⁹ a scale that shall be applied to the scientific literature discussed herein. Thereafter, we place accidental injuries and bleeding disorders under the evidence-based microscope, examining that literature in light of the Oxford rating scale for evidence-based medicine. We then detail the current state of knowledge on biomechanics and

¹⁶ *Id.* at 576-83.

¹⁷ *Id.* at 628-29.

¹⁸ See generally L. Frasier et al., ABUSIVE HEAD TRAUMA IN INFANTS & CHILDREN: A MEDICAL, LEGAL, AND FORENSIC REFERENCE (2006); L. Rorke-Adams et al., *Head Trauma*, in CHILD ABUSE: MEDICAL DIAGNOSIS & MANAGEMENT 53-119 (R. Reece & C. Christian eds. 2009); C. Jenny, CHILD ABUSE AND NEGLECT: DIAGNOSIS, TREATMENT, AND EVIDENCE, ch. 6, 39-48 (2010).

¹⁹ See OXFORD CENTRE FOR EVIDENCE-BASED MEDICINE, *Levels of Evidence* (March 2009), www.cebm.net/index.aspx?o=1025 (last updated Jan. 29, 2013)(last visited March 1, 2013).

hypoxia-ischemia,²⁰ highlighting the weaknesses and limitations that infect that literature. Finally, we magnify the microscopic examination of the differential diagnosis method, examining it closely with both a scientific and legal lens. In so doing, we garner and dissect the scientific and legal arguments around that methodology, discuss the fallacious arguments critiquing the AHT/SBS literature, and propose some solutions going forward for the identification and promulgation of sound scientific evidence on the topic in the legal setting.

Evidence-Based Medicine & AHT/SBS

“I look upon it as being a great part of the art to be able to judge properly of that which has been written.”²¹

-Hippocrates

Physicians have been trained in the natural sciences, the advancement of medical knowledge, and the critical appraisal of medical literature since the dawn of medicine.²² Medical journals and the peer review system now date back nearly two hundred years.²³ With the burgeoning of medical publications and the advent of electronic indexing of the medical literature in the 1970s and 1980s, it clearly became untenable for an individual practitioner to remain aware of all the research activity in even a small specialty of medical practice. From this environment of rapid scientific discovery grew a new movement: Evidence-Based Medicine (EBM). Perhaps described best by one of its founders, Dr. David Sackett, EBM is the “conscientious, explicit, and judicious use of the *current, best* evidence in making decisions about individual care.”²⁴ The review article containing an expert’s opinion about research was replaced by a systematic review: a thorough, exacting, and repeatable methodology for grading and summarizing the most current medical evidence.

²⁰ “Hypoxia” is defined as low tissue oxygenation. See Christian Rosenberger et al., *Immunohistochemical Detection of Hypoxia-Inducible Factor-1 α in Human Renal Allograft Biopsies*, 18 J. AM. SOC. NEPHROLOGY 343, 349 (2006); “Ischemia” is defined as a deprivation of blood supply to body tissue. See, e.g., David J. Hearse, *Ischemia, Reperfusion, and the Determinants of Tissue Injury*, 4 CARDIOVASCULAR DRUGS & THERAPY 767, 768 (1990).

²¹ Hippocrates, *OF THE EPIDEMICS*, (Francis Adams trans., Univ. of Adelaide Library) (2007), available at <http://ebooks.adelaide.edu.au/h/hippocrates/edpidemics/> (last updated Sept. 16, 2012; last visited March 1, 2013).

²² See, e.g., *id.*

²³ THE LANCET, a prominent medical journal, was first published October 5, 1823. See *About the Lancet Medical Journal*, ELSEVIER, INC., <http://www.thelancet.com/lancet-about>.

²⁴ David L. Sackett et al., *Evidence Based Medicine: What It Is and What It Isn’t*, 312 BMJ 71, 71 (1996) (emphasis added).

A central tenet of EBM is that not all evidence is of equal quality.²⁵ In seeking out the “best” evidence, one has to make value judgments, and do so without reference to the outcome of the study.²⁶ The value judgments that ascribe one study as better than another are primarily based upon the technical elements of the design and execution of the study.²⁷ There has been a proliferation of “rating scales” that rate medical studies.²⁸ Some scales²⁹ are more appropriate for assessing the quality of literature on *therapeutic* modalities in clinical medicine; others provide multiple scales that are amenable to different kinds of studies.³⁰ For example, the Center for Evidence Based Medicine (CEBM) at The University of Oxford utilizes five different scales—for assessments of literature on therapy, prognosis, *diagnosis*, symptom prevalence, and economic decision analysis.³¹ The CEBM scale for diagnostic literature is listed below in Table 1.³²

Table 1 Centre for Evidence Based Medicine Levels of Evidence Scale for a Diagnosis³³

a	Systematic review (with homogeneity) of level 1 diagnostic studies or clinical decision rule with 1b studies from different clinical centers.
	Validating cohort study with good reference standards or clinical decision rule

²⁵ See, e.g., Brad Petrisor & Mohit Bhandari, *The Hierarchy of Evidence: Levels and Grades of Recommendation*, 41 INDIAN J. ORTHOPAEDICS 11, 11 (2007).

²⁶ See Sackett et al., *supra* note 23, at 72.

²⁷ See Petrisor & Bhandari, *supra* note 24, at 11-12.

²⁸ See, e.g., David Moher et al., *Preferred Reporting Items for Systematic Reviews and Meta-Analysis: The PRISMA Statement*, 6 PLOS MED. 1, 4-5 (2009), available at

<http://www.plosmedicine.org/article/info%3Adoi%2F10.1371%2Fjournal.pmed.1000097#s4> (systemic reviews and meta analysis); Kenneth F. Schulz, *CONSORT 2010 Statement: Updated Guidelines for Reporting Parallel Group Randomized Trials*, 152 ANNALS OF INTERNAL MED. 726, 727 (2010), available at

<http://annals.org/article.aspx?articleid=746833>; Erik von Elm et al., *The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: Guidelines for Reporting Observational Studies*, 61 J. CLINICAL EPIDEMIOLOGY 344, 346-47 (2008), available at

<http://download.journals.elsevierhealth.com/pdfs/journals/0895-4356/PIIS0895435607004362.pdf> (observational studies including cohort, case-control, and cross-sectional studies).

²⁹ This was the type of scale utilized by Dr. Donohoe in *Mark Donohoe. Evidence-Based Medicine and Shaken Baby Syndrome Part 1: Literature Review*, 24 AM. J. OF MED. PATHOLOGY 239, 240-41 (2003), and was heavily relied upon by the dissenting justices in *Cavazos v. Smith*, as well as by other legal scholars. See *Cavazos*, 132 S. Ct. 2,10 (2011); Tuerkheimer, *supra* note 3, at 12-13; See also Molly Gena, *Shaken Baby Syndrome: Medical Uncertainty Casts Doubt on Convictions*, 3 WIS. L. REV. 701, 706, 710, 727 (2007)

³⁰ See OXFORD CENTRE FOR EVIDENCE-BASED MEDICINE, *supra* note 18.

³¹ *Id.*

³² *Id.*

³³ Bob Phillips et al., *Levels of Evidence (March 2009)*, OXFORD CENTRE FOR EVIDENCE BASED MEDICINE (Aug. 2, 2013), <http://www.cebm.net/index.aspx?o=1025>.

1b	tested within one clinical center.
1c	A diagnostic finding so strong that it absolutely confirms or refutes the diagnosis.
2a	Systematic review (with homogeneity) of 2b or better studies.
2b	Exploratory cohort study with good reference standards; clinical decision rule after derivation or validated only on split-samples or databases.
3a	Systematic review (with homogeneity) of 3b or better studies.
3b	Non-consecutive study or without consistently applied reference standards.
4	Case-control study, poor, or non-independent reference standard.
5	Expert opinion without explicit, critical appraisal; or based on physiology, bench research, or “first principles.”

A prime example of the misapplication of ratings scales is that of Donohoe in his oft-cited, unfortunate article *Evidence-Based Medicine and Shaken Baby Syndrome*.³⁴ While Donohoe decries the absence of randomized-controlled trials,³⁵ the CEBM levels of evidence scale for a diagnosis does not even include randomized-controlled trials.³⁶ The CEBM correctly recognizes that the randomized trial, while excellent for evaluating a therapy, is not an appropriate tool for evaluating a diagnosis.

The last element of the “current, best evidence” in EBM is the emphasis on the adjective “current.”³⁷ Some critics of the AHT/SBS diagnosis propose a level of diagnostic abstinence until some future level of scientific precision (akin to a “DNA-type” evidence) can be achieved.³⁸ Yet, even the most ardent EBM advocates would not purport such diagnostic

³⁴See, e.g., Donohoe, *supra* note 25.

³⁵See Donohoe, *supra* note 25, at 240.

³⁶Bob Phillips et al., *Levels of Evidence (March 2009)*, OXFORD CENTRE FOR EVIDENCE BASED MEDICINE (Aug. 2, 2013), <http://www.cebm.net/index.aspx?o=1025>.

³⁷McClellan et al., *supra* note 23, at v.

³⁸See Findley et al., *supra* note 3, at 305–06.

impotence whilst awaiting some yet-unrealized, “absolute” diagnostic certainty. The emphasis of EBM is to focus on the best *available* evidence, not to discard the evidence we have simply because better evidence is not yet available.

Accidental Injury

Accidents,³⁹ often as a result of falls, are a commonly reported cause of head injury in children.⁴⁰ In Fujiwara et al.’s review of 28 AHT cases and 232 non-abusive head injuries, fall was the history presented in at least 17.9% and 62.9% of the cases, respectively.⁴¹ This section details the evidence that physicians can reliably utilize to distinguish accidental from non-accidental trauma. While it might seem that accidental trauma and inflicted trauma would be difficult to differentiate, a number of studies demonstrate that this actually is possible.

A. Short falls

A particularly common childhood injury reported to pediatricians is the “short fall.”⁴² Because injuries later thought to be abusive are often blamed on short falls, they merit special attention. Authors in pediatric literature have defined short falls as heights varying from less than 15 feet,⁴³ to 10 feet,⁴⁴ to less than 1.5 meters (4.9 feet).⁴⁵ Although there exists no

³⁹Public health advocates, for various reasons, prefer the term “preventable injury.” For the purposes of this paper, and with no intent to diminish the laudable public health reasons for the use of that terminology, we shall use the term “accident,” as it is the more commonly utilized term in the medical literature in comparison to abusive injury.

⁴⁰See Takeo Fujiwara et al., *Characteristics That Distinguish Abusive From Nonabusive Head Trauma Among Young Children Who Underwent Head Computed Tomography in Japan*, 122 PEDIATRICS e841, e843 (October 2008).

⁴¹*Id.* at e842–43.

⁴²Much as all cancers are not the same, all “short falls” are not the same. Stairway falls, falls from moving objects (such as shopping carts or moving strollers or walkers), or falls involving occipital (back of the head) impact do not entail the same biomechanics as simple short falls off furniture (such as beds or couches), where typically the frontal or parietal (side) areas of the skull are impacted. The varying biomechanical forces on different anatomic structures (such as the head, the neck, or the torso) in stairway falls, falls from moving objects, or falls with occipital impact warrant their distinction into a separate category than the “simple short fall.” Hereinafter, unless otherwise stated, the literature reviewed pertains to simple short falls.

⁴³Michael Y. Wang et al., *Injuries From Falls in the Pediatric Population: An Analysis of 729 Cases*, 36 J. PEDIATRIC SURGERY 1528, 1528 (2001); James A. Murray et al., *Pediatric Falls: Is Height a Predictor of Injury and Outcome?*, 66 AM. SURGERY 863, 863 (2000).

⁴⁴John Plunkett, *Fatal Pediatric Head Injuries Caused by Short Distance Falls*, 22 AM. J. FORENSIC MED. PATHOLOGY 1, 10 (2001); Gregory Riber, *Fatal Falls in Childhood*, 14 AM. J. FORENSIC MED. PATHOLOGY 201, XXX (1993).

⁴⁵David L. Chadwick et al., *Annual Risk of Death Resulting From Short Falls Among Young Children: Less Than 1 in 1 Million*, 121 PEDIATRICS 1213, 1213 (2008).

standardized definition of a “short fall,” more recently consensus has shifted toward recognizing a “short fall” as a fall of less than 1.5 m.⁴⁶

There is an abundance of medical literature on pediatric short falls. Studies have focused on a variety of injury aspects—whether there are observed differences in witnessed versus unwitnessed falls, in falls of varying degrees of height, and in falls with varying biomechanical forces and aspects.⁴⁷ Study designs have ranged from isolated case reports to large epidemiologic studies and systematic reviews.⁴⁸ Methodological differences have posed challenges to collectively assimilating data in systematic reviews.⁴⁹ However, a comprehensive review⁵⁰ of the scientific literature does permit the clinician to draw the following conclusions with a reasonable degree of medical certainty.

Scientific Conclusion # 1: When caregivers have been surveyed, they report that short falls are common, but severe⁵¹ injuries from short falls are rare.

In 2010, Suzanne B. Haney et al. surveyed 307 parents, asking if their child had fallen off a “high surface” such as a table, bed, or dresser, before the age of 2.⁵² Forty percent of parents recalled such a fall, and 59% of parents recalled more than one such fall.⁵³ Among the 209 reported falls, the only serious injuries reported were 2 concussions.⁵⁴ There were no reported

⁴⁶See *id.* at 1213.

⁴⁷See, e.g., *id.*

⁴⁸See, e.g., *id.*

⁴⁹See, e.g., *id.* at 1220. Some of these methodological variances have included variations in short fall definition, variations in inclusion and exclusion criteria of patients, and variations in outcome aspects.

⁵⁰This section is not intended to be an exhaustive review of the topic as it is outside the scope and purpose of this paper. For a more thorough review of the topic, see CHILD ABUSE AND NEGLECT: DIAGNOSIS, TREATMENT, AND EVIDENCE 39–48 (C. Jenny ed., 2010); ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN: A MEDICAL, LEGAL, AND FORENSIC REFERENCE (L. Frasier et al. eds., 2006); CHILD ABUSE MEDICAL DIAGNOSIS AND MANAGEMENT 53–119 (Robert M. Reece et al. eds., 3d ed. 2009).

⁵¹Hereinafter, the use of the terms “severe injury” or “serious injury” refer to their meaning within the AIS (Abbreviated Injury Scale). See *Abbreviated Injury Scale*, ASSOCIATION FOR THE ADVANCEMENT OF AUTOMOTIVE MEDICINE (Oct. 28, 2012), <http://www.aaam1.org/ais/>; *Abbreviated Injury Scale*, TRAUMA.ORG (Oct. 28, 2012), <http://www.trauma.org/archive/scores/ais.html>. Although it is common, clinically, to incorporate the AIS score into an Injury Severity Score (ISS) when assessing overall trauma to the human body, for the purposes of this article, “severe injury” or “serious injury” will refer to their use within the AIS. Although the AIS dictionary has specific codes for specific head injuries, typically intracranial hemorrhages have scores of either 3 or 4 (3 = serious; 4 = severe) and cerebral edema (brain swelling) has a score of 5 (critical). See Thomas Songer, *Measuring Injury Severity* (Oct. 28, 2012), available at <http://www.pitt.edu/~epi2670/severity/severity.pdf>.

⁵²Suzanne B. Haney et al., *Characteristics of Falls and Risk of Injury in Children Younger Than 2 Years*, 26 PEDIATRIC EMERGENCY CARE 914, 915 (2010).

⁵³*Id.* at 915.

⁵⁴*Id.* at 917.

subdural hematomas, retinal hemorrhages, or deaths.⁵⁵ These results accorded with Warrington et al., a similar, but much larger, study from the UK in 2001.⁵⁶ In that study, when their child had reached six months of age, parents were asked to describe any accident that had occurred with their child since birth.⁵⁷ The authors received data on over 2500 children, with over 3300 falls being reported.⁵⁸ Of these approximate 3300 falls, 1782 (53%) were falls from beds or settees.⁵⁹ Only 21 falls (less than 1%) resulted in concussion or fracture.⁶⁰ There were no reported intracranial injuries or death.⁶¹ Other authors who have surveyed parents/caregivers have obtained similar results.⁶² Although these two cross-sectional studies would merit only a 4 on the Oxford CEBM scale,⁶³ they hold some scientific significance in that the subjects studied had no motivation or inclination to provide inaccurate data.⁶⁴

Scientific Conclusion #2: Short falls occurring in objective settings, such as hospitals, have not resulted in subdural hematoma or death.

In 1993, Lyons and Oates reported on 207 children who fell out of bed in the hospital in which a nurse either observed the fall or attended to the child within seconds of the fall.⁶⁵ Falls ranged from 32–54 inches.⁶⁶ One child sustained a skull fracture and another child sustained a clavicle fracture.⁶⁷ There were no multiple injuries, visceral injuries, severe head injuries, or deaths.⁶⁸ These results accorded not only with prior studies by Nimityongskul⁶⁹ in 1987 and

⁵⁵*Id.* at 918.

⁵⁶S.A. Warrington et al., *Accidents and Resulting Injuries in Premobile Infants: Data From the ALSPAC Study*, ARCH. DIS. CHILD. 104, 104 (2001).

⁵⁷*Id.* at 105.

⁵⁸*Id.* at 104.

⁵⁹*Id.* at 105.

⁶⁰*Id.*

⁶¹*Id.* at 106-07.

⁶²See Harvey Kravitz et al., *Accidental Falls from Elevated Surfaces in Infants from Birth to One Year of Age*, 44 PEDIATRICS 869 (1969); Ray E. Helfer et al., *Injuries Resulting from when Small Children Fall Out of Bed*, 60 PEDIATRICS 533 (1977).

⁶³See Centre for Evidence Based Medicine, *supra* note 26.

⁶⁴Certainly, a limitation of the study was that the data was subject to recall inaccuracy or bias. But, there was little concern for intentionally skewed data (as is a concern in suspected AHT cases).

⁶⁵Thomas J. Lyons & R. Kim Oates, *Falling Out of Bed: A Relatively Benign Occurrence*, 92 PEDIATRICS 125 (1993).

⁶⁶*Id.* at 126.

⁶⁷*Id.*

⁶⁸*Id.*

⁶⁹Nimityongskul P, Anderson LD, *The Likelihood of Injuries When Children Fall Out of Bed*, 7(2) J. PEDIATRIC ORTHOPEDICS 184, 184–186 (1987).

Levene⁷⁰ in 1991, but with subsequent studies by Monson⁷¹ in 2008, Ruddick⁷² in 2010, and Schaffer⁷³ in 2012. In total, these case series describe over 620 falls in objective⁷⁴ settings (i.e., hospitals), with no consequent serious head injuries or deaths. Although these studies are case series, or level 3b evidence on the CEBM scale, given the increased level of objectivity to the observed injuries, they warrant significant scientific consideration.

Scientific Conclusion #3: Children in large, licensed daycares rarely die from short falls.

Another source of independently observed childhood falls is daycare. Licensed daycare centers are an attractive environment to study short falls because structural layout and institutional policy effectively minimizes abuse and facilitates reporting of accidents and abuse.⁷⁵

Chadwick conducted a comprehensive review of all daycare studies in the world's medical literature.⁷⁶ He found 25 studies that focused on injuries occurring in daycare, studies from the US, Canada, Sweden, Norway, and Denmark.⁷⁷ Only 2 of the 25 studies specifically looked at deaths in the daycare setting. Good et al. reviewed data on over 520,000 children in daycare centers and found no deaths attributed to falls.⁷⁸ Wrigley and Dreby examined data on over 6 million children in child care centers over an 18 year period (1985–2003) and found only

⁷⁰S. Levene & G. Bonfield, *Accidents on Hospital Wards*, 66(9) ARCHIVES OF DISABLED CHILDREN 1047, 1047–1049 (1991).

⁷¹S. Monson et al., *In Hospital Falls of Newborn Infants: Data From a Multihospital Healthcare System.*, 122 PEDIATRICS e 227, e277–e280 (2008).

⁷²C. Ruddick et al., *Head Trauma Outcomes of Verifiable Falls in Newborn Babies*, 95(2) ARCHIVES OF DISABLED CHILDREN FETAL NEONATAL ED. F144, F144-5 (2010).

⁷³P.L. Schaffer et al., *Pediatric Inpatient Falls and Injuries: A Descriptive Analysis of Risk Factors*, J. SPECIAL 17(1) PEDIATRIC NURSING 10-8, 10-8 (2012).

⁷⁴One author has asserted that hospital settings may not be “objective,” as there is an equal likelihood for malicious acts by nursing or other staff in hospitals as in any other setting. See J. Ehsani et al., *The Role of Epidemiology in Determining if a Simple Short Fall Can Cause Fatal Head Injury in an Infant*, 31 AM. J. FORENSIC PATHOLOGY 287, 287-298 (2010). However, hospital settings are by nature, less private settings, with clear understanding to all within those settings of a diminished level of privacy. Given that abusive acts occur in private settings, it is unreasonable to assume an equal likelihood of abuse in inherently less private/more public settings.

⁷⁵Julia Wrigley & Joanna Dreby, *Fatalities and the Organization of Child Care in the United States*, 70(5) AM. SOCIOLOGY REV. 729, 729-75 (2005).

⁷⁶See Chadwick, *supra* note 38.

⁷⁷*Id.*

⁷⁸Good et al's review found 9 deaths in child care centers, but 6 were attributed to natural causes, and 3 to “unintentional” causes (2 asphyxiation and 1 pedestrian/MVA). See Susan E. Good et al., *Children's Deaths at Day-care Facilities*. PEDIATRICS, 1039, 1039-41 (1994).

2 reported deaths attributed to falls in large, licensed child care centers.⁷⁹ However, these two reported deaths stemmed from newspaper reports and, consequently, provided no detailed data for scientific analysis.⁸⁰ Despite having over 900,000 children under age 2 in US day care centers,⁸¹ with multiple falls occurring daily, to date there is no peer-reviewed, medical report of a death resulting from a short fall in a large, licensed day care center.⁸²

Scientific Conclusion #4: Severe injuries and deaths are rare in short falls witnessed by two or more adults, but ironically, are more common in short falls witnessed by a single adult.

Williams⁸³ reviewed the cases of 398 patients treated with injuries resulting from a fall. Of these 398 patients, 106 were less than 3 years old and the fall was witnessed by at least two adults.⁸⁴ The only death in this group was a child who fell 70 feet.⁸⁵ The only serious injuries resulting from observed falls less than 10 feet were children (3%) with depressed skull fractures after falling against an “edged” surface.⁸⁶ In contrast, among 53 children under 3 whose falls were witnessed by fewer than two adults, 18 had serious injuries and 2 died after reported falls of less than 5 feet.⁸⁷ Similar results and conclusions were reached by Wrigley and Dreby⁸⁸ in their comprehensive review of daycare literature, by Reece & Sege⁸⁹ and by Johnson⁹⁰ et al. Although Williams’s, Johnson’s, and Reece & Sege’s case series only merit level 3b evidence on the CEBM scale, they provide interesting insight into the histories provided by caregivers in abusive and accidental injuries.

⁷⁹See Wrigley & Dreby, *supra* note 64, at 749.

⁸⁰See Chadwick, *supra* note 38, at 1216.

⁸¹US Department of Education, *Child Care and Early Education Arrangements in Infants, Toddlers, and Preschoolers: 2001* (2001), available online at <http://nces.ed.gov/pubs2006/2006039.pdf>, last visited 9/5/2012.

⁸²See Chadwick, *supra* note 38.

⁸³R.A. Williams, *Injuries in Infants and Small Children Resulting from Witnessed and Corroborated Free Falls*, 31(1) J. TRAUMA 1350, 1350 (1991).

⁸⁴*Id.* at 1351.

⁸⁵*Id.*

⁸⁶*Id.*

⁸⁷*Id.*

⁸⁸See Wrigley & Dreby, *supra* note 64.

⁸⁹R.M. Reece & R. Sege, *Childhood Head Injuries: Accidental or Inflicted?*, 154 ARCHIVE PEDIATRIC ADOLESCENT MEDICINE 11, 11 (2000).

⁹⁰K. Johnson et al., *Accidental Head Injuries in Children Under 5 Years of Age*, 60 CLINICAL RADIOLOGY 464, 464 (2005).

Scientific Conclusion #5: If reports of deaths from uncorroborated short falls are accepted as valid, then short falls appear to be more dangerous than longer falls.

Williams noted another anomaly. In his study of 398 patients, uncorroborated reports indicated 2 deaths followed falls of less than 5 feet, but no deaths followed falls of 6–11 feet or 12–23 feet.⁹¹ Chadwick et al. noted similar findings in his review of 317 uncorroborated falls requiring medical attention.⁹² Among 183 children who reportedly fell 5–45 feet, only 1⁹³ died. Among 100 children reported to have fallen less than 4 feet, 7 died.⁹⁴ All 7 children had other factors concerning for a false history, including old fractures, bruising on the trunk or extremities, genital injury, or more than one impact point on the head.⁹⁵ Similar results were obtained by Reece & Sege.⁹⁶ Although the biomechanics of a short fall are a complex phenomenon, and probably not simply a factor of height,⁹⁷ other authors have also noted a correlation between increased fall height and injury severity.⁹⁸

Scientific Conclusion # 6: Well-designed, prospective studies reveal that severe injuries or death resulting from short falls are rare events.

In 1992, Duhaime et al.⁹⁹ prospectively studied 100 patients less than 2 years of age who suffered head injuries. In efforts to avoid “circularity” concerns, Duhaime et al. used strict criteria for determining “inflicted” injury. The authors excluded retinal hemorrhages (RHs) as a diagnostic criterion and only included SDHs that had no history of trauma but had clinical or radiologic findings of blunt impact to the head. Thus, the authors designed an algorithm that was

⁹¹ See Williams, *supra* note 72, at 1351.

⁹² D.L. Chadwick et al., *Deaths from Falls in Children: How Far is Fatal?*, 31 J. TRAUMA 1353, 1353–55 (1991).

⁹³ *Id.* at 1354. An 11 month old child was found dead beneath a second story window. Parents were not aware of the child’s absence until notified by neighbors who found the child.

⁹⁴ *Id.* at 1354.

⁹⁵ *Id.* at 1353.

⁹⁶ See Reece & Sege, *supra* note 78, at 11.

⁹⁷ See N.G. Ibrahim et al., *Influence of Age and Fall Type on Head Injuries in Infants and Toddlers*, 30 INT’L J. DEVELOPMENTAL NEUROSCIENCE 201, 201–206 (2012) (proposing that the fall type and age of child are important factors in the resulting injury of a child); see also James A. Murray et al., *Pediatric Falls: Is Height a Predictor of Injury and Outcome?*, 66 AM. SURGEON 863, 863–865 (2000).

⁹⁸ See Angela K. Thompson et al., *Pediatric Short-Distance Household Falls: Biomechanics and Associated Injury Severity*, 43 ACCIDENT ANALYSIS PREVENTION 143, 143 (2011); see also Johnson et al., *supra* note 49, at 467; Michael Y. Wang et al., *Injuries from Falls in the Pediatric Population: An Analysis of 729 Case*, 36 J. PEDIATRIC SURGERY 1528, 1528 (2000).

⁹⁹ A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age*, 90 PEDIATRICS 179, 179 (1992).

“deliberately biased to reduce false positives and, thus, underestimate the true incidence of child abuse.”¹⁰⁰ In the Duhaime et al. cohort, 76 patients’ injuries were determined to be from accidental causes, and 24 were determined to be inflicted.¹⁰¹

Of the 100 studied patients, 73 suffered head injuries from reported falls, 9 from motor vehicle accidents, 2 from impacts by other objects, 2 admitted assaults, and 14 without any history.¹⁰² Of those 73 reported falls, 34 (47%) were from falls less than 4 feet, 21 (29%) from falls greater than 4 feet, and 18 (25%) from falls from walkers or down stairs.¹⁰³ Of the 34 reported short falls, 26 were determined to be accidental, and 8 were determined to be inflicted. Of the 26 accidental short falls, none had SDHs or RHs.¹⁰⁴ In Duhaime et al.’s entire 100-patient cohort, there were only 4 deaths—3 from the inflicted group and 1 from the accidental group.¹⁰⁵ The only death from the accidental category was a passenger in a high-speed motor vehicle accident.¹⁰⁶ Similar results were obtained by Bechtel et al.¹⁰⁷ in their prospective study of 87 children aged 0–2 years at Yale Children’s Hospital from 2000–2002.

In 2011 Thompson et al.¹⁰⁸ reported their prospective study of 79 children less than 4 years of age who presented to the emergency department of Kosair Childrens’ Hospital (Louisville, KY) between May 2008–July 2009 with a complaint of a household fall from a bed, sofa, or similar furniture. The authors sought to determine the severity of injuries that resulted from accidental short-distance household falls in children and to investigate the association of fall environment and biomechanical measures with injury outcomes.¹⁰⁹ The authors excluded all children suspected of abuse and included only children that were “definite” or “likely” accidents.¹¹⁰ The authors conducted interviews with the caregivers and in-depth scene

¹⁰⁰ *Id.* at 180.

¹⁰¹ *Id.*

¹⁰² *Id.* at 181.

¹⁰³ *Id.*

¹⁰⁴ *Id.* Three of the 26 short falls had epidural hemorrhages (EDH’s). Of the 21 longer falls, 6 had very focal subarachnoid hemorrhages or brain contusions. All fall children had benign hospital courses.

¹⁰⁵ *Id.* at 181-182.

¹⁰⁶ *Id.*

¹⁰⁷ Kirsten Bechtel et al., *Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma*, 114 PEDIATRICS

¹⁰⁸ See Thompson et al., *supra* note 87, at 144.

¹⁰⁹ *Id.* at 143.

¹¹⁰ *Id.* at 144.

investigations in all 79 cases in order to obtain information regarding fall dynamics and to determine biomechanical measures associated with these falls.¹¹¹

Of the 79 subjects enrolled, 15 had no injuries, 45 had minor (AIS 1) injuries,¹¹² 17 had moderate (AIS 2) injuries,¹¹³ and 2 had serious (AIS 3) injuries.¹¹⁴ No subjects had injuries classified as AIS 4 or higher, and there were no fatalities.¹¹⁵ The authors also determined that, in their study, “furniture height, impact velocity and child BMI were found to have the greatest influence on injury severity outcomes.”¹¹⁶ Children with moderate or serious injuries tended to have fallen from greater heights, had greater impact velocities, and had a lower BMI than those with minor or no injuries.¹¹⁷ Thompson et al. concluded that “this study provides a comprehensive evaluation of the biomechanics of short-distance household falls and investigated the association of biomechanical and fall environment measures with injury severity.”¹¹⁸ Children aged 0–4 years involved in a short-distance household fall did not sustain severe or life-threatening injuries.¹¹⁹ Duhaime’s, Bechtel’s, and Thompson’s studies merit level 2b evidence on the CEBM scale.

Scientific Conclusion # 7: Systematic reviews of the short fall literature indicate that short falls rarely cause death in children.

In 2009, Chadwick et al. sought to numerically quantify the risk of death from short falls.¹²⁰ The authors performed an *extensive* review of the published medical literature on short falls, “including 5 book chapters, 2 medical society statements, 7 major literature reviews, 3 public injury databases, and 177 peer-reviewed, published articles indexed in the National Library of Medicine.”¹²¹ The authors examined data and literature from any and every short fall aspect: reliably witnessed falls, child-care studies, studies of large clinical populations (more than fifty cases), studies of single cases or small series (less than fifty cases), studies of long

¹¹¹ *Id.*

¹¹² *Id.* at 145. These were described as “lacerations” or “contusions.”

¹¹³ *Id.* These were described as “fractures.”

¹¹⁴ *Id.* These were described as “small isolated SDH’s.”

¹¹⁵ *Id.* at 143.

¹¹⁶ *Id.*

¹¹⁷ *Id.* at 149.

¹¹⁸ *Id.*

¹¹⁹ *Id.*

¹²⁰ See Chadwick, *supra* note 38, at 1213.

¹²¹ *Id.*

falls, pathologic and cadaveric studies, studies involving biomechanical analysis, playground fall studies, studies involving falls down stairs, walker-related falls, parent-observation studies, and studies specifically addressing short-fall death.¹²²

When reviewing the two large injury databases (California’s EPIC¹²³ database and the CDC’s WISQRS¹²⁴ database), Chadwick et al. found that WISQRS allowed determination of the total fall death rate for children less than four years of age, but did not provide stratification according to fall type/height.¹²⁵ Per the WISQRS database, “[t]he ‘all fall’ death rate was 3 cases per 1 million young children per year.”¹²⁶ However, California’s EPIC database did stratify data for short falls.¹²⁷ For the period 1999–2003, the EPIC database revealed 6 short fall fatalities.¹²⁸ In a state with 2.5 million children under 5 years old, this calculated to 0.48 deaths per million children per year.¹²⁹ Chadwick et al. provided comparative risk estimates for other conditions that caused death in infants and children (see Figure 1 below):

TABLE 9 Some Specific Conditions Causing Death in Infants and Young Children in California

Condition	Deaths per 1 Million Young Children per y
Prematurity (all deaths recorded in first year of life)	165
Congenital malformations	316
Neoplasms	33
Respiratory diseases	38
Accidents	121
Short Falls	0.48
Homicide ^a	22

Figure 1: Table from David L. Chadwick et al., *Annual Risk of Death Resulting*

From Short Falls Among Young Children: Less than 1 in 1 Million, 121 PEDIATRICS 1213, 1220

(2008). Reprinted with permission.

Hence, based upon Chadwick et al.’s data, the chance of any given child dying of a short fall in any given year is approximately one in two million. However, to truly approximate the

¹²² *Id.*

¹²³ *Id.* at 1214. (“EPIC” stands for Epidemiology and Prevention for Injury Control Branch.)

¹²⁴ *Id.* (“WISQRS” stands for Web-based Injury Statistics Query and Reporting System.)

¹²⁵ *Id.*

¹²⁶ *Id.*

¹²⁷ *Id.*

¹²⁸ *Id.*

¹²⁹ *Id.*

probability of a single fall causing death, one would need to multiply the one in two million by the number of falls a typical toddler or child experiences in a year.

Ehsani et al. also conducted a comprehensive review of the short fall literature with the aim of answering the question: “Can a simple short fall cause fatal head injury in an infant?”¹³⁰ Toward this aim, Ehsani et al. considered 1055 publications for inclusion.¹³¹ Using explicit selection criteria, only 27 publications were included in their review.¹³² The authors concluded that it is “rare, but possible, for fatal head injury to occur from a simple short fall.”¹³³ The authors went on to state that, “[l]arge population studies of childhood injuries indicate that severe head injury from a short fall is extremely rare. This is counter pointed by a single documented case report that demonstrates it can happen.”¹³⁴

Plunkett also sought to assess the plausibility of short fall deaths.¹³⁵ He reviewed the National Electronic Injury Surveillance System (NEISS) and determined that, over a 12 year period (1988–1999), 18 children had fallen from playground equipment and subsequently died.¹³⁶ Plunkett’s data, however, suffer from several limitations. First, Plunkett’s study is not a true systematic review of the short fall literature. Second, the NEISS database Plunkett reviewed suffers from selectivity bias.¹³⁷ Finally, Plunkett’s reported deaths are inaccurate.¹³⁸

The Chadwick et al. systematic review of the short fall literature constitutes level 2a evidence on the CEBM scale, whereas Plunkett’s study constitutes level 3b or 4 evidence.

¹³⁰Johnathon P. Ehsani et al., *The Role of Epidemiology in Determining if a Simple Short Fall can Cause Fatal Head Injury in an Infant*, 31 AM. J. FORENSIC MED. PATHOLOGY 287, 287 (2010).

¹³¹*Id.*

¹³²*Id.*

¹³³*Id.*

¹³⁴*Id.* The single case alluded to by the authors is a reported videotaped short fall resulting in death after a brief lucid interval in Plunkett’s review of the US Consumer Product Safety Commission database. *See* Plunkett, *supra* note 36, at 4; Ehsani, *supra* note 115, at 290.

¹³⁵*See* Plunkett, *supra* note 36, at 1.

¹³⁶*Id.* at 2.

¹³⁷*Id.* Since the database is geared towards detection of fatalities related to products, it can miss fatalities not related to products, and is not designed to discriminate false histories related to products (probably including abusive injuries within the data subset).

¹³⁸*See* Chadwick, *supra* note 38, at 1215. “Nine of the 18 children who died were [older than] 5 years of age. Among the 9 young children, 4 cases were not witnessed at all, even by other children. Of the remaining 5 cases, 1 fall height was estimated at [greater than] 2.0 m. Of the remaining 4 cases, 1 had no autopsy, and the cause of death in that case was uncertain . . . With the determination that 3 of the cases were valid, the annual population risk for a short-fall death of a young child in this (playground) sample can be calculated as 3 fatalities/(400 000 x 12) = 0.625 cases per 1 million young children per year.” *Id.*

B. Other accident literature

In addition to the short-fall literature, several well-designed, prospective studies, comparing accidents and abuse cases, have identified clinical variables that can discriminate accidents from abuse case with a high degree of statistical significance. The strength of these studies lies in their ability to validate prior exploratory studies with good reference standards, thus *constituting level 1b evidence* on the CEBM scale. For example, Vinchon et al.'s 2010 prospective series of 84 patients who sustained injuries from either witnessed accidents (N=39) or confessed inflicted head injury (N=45—obtained from judicial sources) determined the specificity and positive predictive value of severe RHs for abusive injury to be 97% and 96%, respectively.¹³⁹ This validated Vinchon et al.'s and Bechtel et al.'s prior exploratory prospective studies in 2005 and 2004, respectively, where the authors found high statistical significance and specificity of more severe RHs for abuse.¹⁴⁰

In enhancing prior research efforts, Hymel et al. developed a national, multi-site research collaborative, Pediatric Brain Injury Research Network (PediBIRN), that is dedicated to conducting rigorous clinical research on pediatric traumatic brain injury.¹⁴¹ The strengths of the studies produced by this collaboration include the prospective multicenter design, the breadth and depth of data capture, and the *a priori* application of criteria for abusive and non-abusive causes (criteria that are specifically designed to minimize circular reasoning and inherent biases).¹⁴² In 2010, the PediBIRN group published the results of their prospective multicenter study that examined the diagnostic, prognostic, and forensic significance of depth of intracranial injury in accidental and non-accidental cases.¹⁴³ After thoroughly reviewing data on 54 children less than 3 years old at 9 sites, the authors found that children “with subcortical injuries (i.e., injuries deeper in the brain) more frequently had been abused” (odds ratio [OR]: 35.6; *P* value <0.001) than had suffered accidents.¹⁴⁴ Although this particular study constitutes level 2b

¹³⁹Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases*, 26 *CHILDS NERVOUS SYS.* 637, 642 (2010).

¹⁴⁰See Matthieu Vinchon et al., *Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study*, 102 *J. NEUROSURGERY* 380, 380 (Supp. 2005); Bechtel, *supra* note 96, at 165.

¹⁴¹Kent P. Hymel et al., *Head Injury Depth as an Indicator of Causes and Mechanisms*, 125 *PEDIATRICS* 712, 713 (Supp. 2010).

¹⁴²*Id.*

¹⁴³*Id.* at 712-13.

¹⁴⁴*Id.* at 712. The odds ratio is quite notable in this study. It indicates that the finding of subcortical brain injury is thirty five times more likely to be the result of abuse than accident. The authors also found that subcortical injury

evidence on the CEBM scale, its rigorous methodology has provided the foundation for the development of emerging higher levels of evidence—clinical prediction rules.¹⁴⁵

Finally, in 2009, Maguire et al. completed a systematic review of the world’s medical literature and identified clinical features that differentiate accidental from non-accidental head injury in children.¹⁴⁶ The authors conducted “[an] all language literature search of 20 electronic databases, websites, references, and bibliographies from 1970–2008.”¹⁴⁷ Using over 100 keyword combinations, this yielded 320 studies for review.¹⁴⁸ Applying strict inclusion and exclusion criteria,¹⁴⁹ the authors determined that 14 studies were appropriate for inclusion, which represented 1655 children: 779 with inflicted brain injury (iBI) and 876 with non-inflicted brain injury (niBI).¹⁵⁰ The authors utilized multilevel logistic regression analysis to arrive at positive predictive values and odds ratios for various clinical features.¹⁵¹

As a result, the authors found that “*apnoea appears to be a critical distinguishing feature* (PPV for abuse 93%, OR 17.06).”¹⁵² This means that, in the comparative diagnostic subset of accidental versus non-accidental injury, a child who arrives at the hospital not breathing is 17 times more likely to have been abused. Likewise, the authors found retinal hemorrhages “were strongly associated” with inflicted brain injury, with a PPV of 71% and an OR of 3.5.¹⁵³ The authors stated, “[a] *child with an intracranial injury who has co-existent retinal haemorrhages [sic] is significantly more likely to have iBI than niBI.*”¹⁵⁴ The authors concluded, “[t]his review is the largest of its kind, and offers for the first time a valid statistical probability of iBI when certain key features are present (e.g. retinal haemorrhages).”¹⁵⁵ Maguire et al.’s systematic review comprises level 2a evidence on the CEBM scale.

(i.e. deeper brain injury) more frequently demonstrated inertial injury and manifested acute respiratory or circulatory compromise. *Id.* These findings had high statistical significance. *Id.*

¹⁴⁵Kent P. Hymel et al., *Derivation of a Clinical Prediction Rule for Pediatric Abusive Head Trauma*, 14 PEDIATRIC CRITICAL CARE MED. (forthcoming 2013) (manuscript at 1) (on file with author).

¹⁴⁶S. Maguire et al., *What Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review*, 94 ARCHIVES DISEASE CHILDHOOD 860, 860 (2009).

¹⁴⁷*Id.*

¹⁴⁸*Id.* at 861.

¹⁴⁹*Id.* at 861, 864.

¹⁵⁰*Id.* at 864.

¹⁵¹*Id.* at 860.

¹⁵²*Id.* at 866. “Apnoea” refers to the cessation of breathing (usually defined as longer than 20 seconds in infants) need a citation (emphasis added).

¹⁵³*Id.* at 865.

¹⁵⁴*Id.* (emphasis added).

¹⁵⁵*Id.*

C. Conclusion

The scientific literature on pediatric accidental injury is robust, sound, and constantly improving. No longer is the isolated case report¹⁵⁶ (level 4 evidence) or the poorly designed case series¹⁵⁷ (level 4 evidence) a sufficient basis for scientific conclusions. Currently, there is level 1b and 2a evidence supporting clinicians in the distinction of abusive from accidental injury when certain clinical features, such as apnea or extensive/severe retinal hemorrhages, are present. And physicians continue to build upon these data, with hopes of soon attaining the highest level of evidence attainable—level 1a. In a recent analysis, Maguire et al. estimated the probability of AHT based on six clinical features, reporting that combinations of the above factors are even more predictive.¹⁵⁸ For example, the author's determined that a child (less than 3 years old) with a subdural hematoma plus any 3 of the following factors: apnea; retinal hemorrhage; rib, skull, or long-bone fractures; seizures; or, head or neck bruising; had a positive predictive value for AHT of greater than 85% and an odds ratio of greater than 100.¹⁵⁹ Armed with such data and analysis, physicians today can confidently conclude that certain infants' injuries are the result of intentional injury, and not the result of accident. And they do so on the basis of the highest quality medical evidence.

Bleeding Disorders

Bleeding disorders may be proposed as the underlying cause for clinical findings in cases of suspected abusive head trauma (AHT). Unlike some hypothesized “mimics” of AHT,¹⁶⁰ bleeding disorders are known causes of intracranial hemorrhage (ICH) and retinal hemorrhage,

¹⁵⁶See generally Scott Denton & Darinka Mileusnic, *Delayed Sudden Death in an Infant Following an Accidental Fall: A Case Report with Review of the Literature*, 24 AM. J. FORENSIC MED. & PATHOLOGY 371 (2003); Horace B. Gardner, *A Witnessed Short Fall Mimicking Presumed Shaken Baby Syndrome (Inflicted Childhood Neurotrauma)*, 43 PEDIATRIC NEUROSURG LANTZ & DANIEL E. COUTURE, *Fatal Acute Intracranial Injury, Subdural Hematoma, and Retinal Hemorrhages Caused by Stairway Fall*, 56 J. FORENSIC SCI. 1648 (2011); Patrick D. Barnes et al., *Traumatic Spinal Cord Injury: Accidental Versus Nonaccidental Injury*, 15 SEMINARS IN PEDIATRIC NEUROLOGY 178 (2008).

¹⁵⁷See generally Jan E. Leestma, *Case Analysis of Brain-Injured Admittedly Shaken Infants: 54 cases, 1969–2001*, 26 AM. J. FORENSIC MED. & PATHOLOGY 199 (2005).

¹⁵⁸Sabine A. Maguire et al., *Estimating the Probability of Abusive Head Trauma: A Pooled Analysis*, J. PEDIATRICS 550 (2011).

¹⁵⁹*Id.* at 550.

¹⁶⁰C.A.B. Clemetson, *Elevated Blood Histamine Caused by Vaccinations and Vitamin C Deficiency May Mimic the Shaken Baby Syndrome*, 62 MED. HYPOTHESIS 533, 534 (2004); Horace B. Gardner, *Immunizations, Retinal and Subdural Hemorrhages: Are They Related?*, 64 MED. HYPOTHESIS 663 (2005).

and are a heterogeneous group of conditions that vary in etiology, presenting symptoms, and prevalence.¹⁶¹ All three of these descriptive characteristics must be considered when evaluating the potential for a bleeding disorder to be the cause of an ICH.

It is important to remember that: 1) *only certain bleeding disorders may cause findings that may be confused with AHT*; 2) *most bleeding disorders are rare*; 3) *the more common bleeding disorders typically are mild*; and 4) *ICH resulting from bleeding disorders is a rare complication of the more severe diseases*.¹⁶² The probability of a rare disorder causing an even rarer manifestation (i.e., ICH) is the main scientific consideration when there is concern for a potential bleeding disorder in a young child with an ICH and a history of no or minimal trauma.¹⁶³

A. Causes (etiologies) of bleeding disorders

Bleeding disorders may be congenital (inherent to the genetic makeup of an individual) or acquired. Congenital bleeding disorders may cause symptoms from the time prior to birth to anytime throughout a person's lifetime.¹⁶⁴ Bleeding symptoms may occur at any time and, depending on the severity of the bleeding disorder, may be asymptomatic (without symptoms) for long periods of time.¹⁶⁵ Acquired bleeding disorders are the result of a condition that is not permanently engendered in a person.¹⁶⁶ As with congenital bleeding disorders, symptoms may present at variable times during one's life, and their severity and duration are dependent upon the specific condition.¹⁶⁷

1. Congenital bleeding disorders

¹⁶¹ Michael O. Gayle et al., *Retinal Hemorrhage in the Young Child: A Review of Etiology, Predisposed Conditions, and Clinical Implications*, 13 EMERGENCY MED. REV. 233 (1995); Lois K. Lee et al., *Intracranial Hemorrhage After Blunt Head Trauma in Children with Bleeding Disorders*, 158 J. PEDIATRICS 1003 (2011).

¹⁶² Carpenter et al., *Technical Report: Evaluating for Suspected Child Abuse: Conditions that Predispose to Bleeding*, PEDIATRICS (In Press).

¹⁶³ *Id.*

¹⁶⁴ S. S. Acharya et al., *Rare Bleeding Disorder Registry: Deficiencies of Factors II, V, VII, X, XII, Fibrinogen and Dysfibrinogenemias*, 2 J. THROMBOSIS & HAEMOSTASIS 248, 249 (2004).

¹⁶⁵ *Id.* at 254-55.

¹⁶⁶ See Monagle & Andrew, *infra* note 147.

¹⁶⁷ Acharya, *supra* note ___, at 249.

Examples of congenital bleeding disorders include hemophilia and von Willebrand disease (VWD). There are many other congenital bleeding disorders,¹⁶⁸ and significant variability exists in the prevalence and presenting symptoms of each of the bleeding disorders, such that each disorder must be considered individually. A discussion of the specific congenital bleeding disorders is outside of the purpose and scope of this review.¹⁶⁹

2. Acquired bleeding disorders

Acquired bleeding disorders may occur at any age, may be isolated, or may occur due to medications, medical illnesses/conditions, or trauma.¹⁷⁰ Specific examples include immune thrombocytopenic purpura (ITP),¹⁷¹ disseminated intravascular coagulation (DIC),¹⁷² vitamin K deficiency bleeding (VKDB),¹⁷³ and liver coagulopathy.¹⁷⁴ Often these acquired bleeding disorders are transient, but some may persist. They either affect platelet number or function or result in a coagulation factor deficiency.

In large part, a clinician can identify acquired causes of bleeding by taking a careful history, performing a detailed physical examination, and ordering the appropriate laboratory tests. For example, a variety of medications can lead to platelet dysfunction (e.g., non-steroidal anti-inflammatory drugs, sodium valproate).¹⁷⁵ A careful medication history can evaluate for this potential.¹⁷⁶ Other acquired bleeding disorders, such as ITP or other causes of

¹⁶⁸ See Carpenter et al., *supra* note 141, at ____.

¹⁶⁹ For a more detailed review of congenital bleeding disorders, see Carpenter et al., *supra*, note 141, at ____.

¹⁷⁰ D. B. Wilson, *Acquired Platelet Defects*, in NATHAN AND OSKI'S HEMATOLOGY OF INFANCY AND CHILDHOOD 1557, ____ (Stuart H. Orkin et al. eds., 6th ed., 2003); P. Monagle & M. Andrew, *Acquired Disorders of Hemostasis*, in NATHAN AND OSKI'S HEMATOLOGY OF INFANCY AND CHILDHOOD 1631, ____ (Stuart H. Orkin et al. eds., 6th ed., 2003).

¹⁷¹ Immune Thrombocytopenic Purpura (ITP) is a condition in which the body forms antibodies to the blood platelets, resulting in their destruction.

¹⁷² Disseminated Intravascular Coagulation (DIC) results from disordered clotting and bleeding, and this can be secondary to a variety of reasons—overwhelming infection, severe trauma, anaphylaxis, etc. It occurs only in children who are severely ill, and may result in bleeding in any part of the body, including intracranial bleeding.

¹⁷³ Vitamin K Deficiency bleeding (previously known as Hemorrhagic Disease of the Newborn) is a bleeding disorder that occurs most commonly in the newborn/early infancy period. Newborns are born with low levels of vitamin K, an essential factor in blood clotting. Unless provided with intramuscular dose of Vitamin K in the newborn period, young infants can suffer bleeding from mucosal surfaces, bleeding from circumcision, generalized bruising, large intramuscular hemorrhages, and intracranial hemorrhage.

¹⁷⁴ “Coagulopathy” can best be defined as a disorder of the natural hemostasis of the body. Since the liver produces some of the clotting factors needed to clot blood, severe liver dysfunction can result in a “coagulopathy.”

¹⁷⁵ Amy A. Hassan & Michael H. Kroll, *Acquired Disorders of Platelet Function*, AM. SOC'Y OF HEMATOLOGY EDUC. PROGRAM BOOK 403, 404-05 (2005).

¹⁷⁶ *Id.* at 403.

thrombocytopenia (low platelet count) can be readily diagnosed on the basis of a lab test—a complete blood count (which manifests the low platelet count).¹⁷⁷ DIC is evident on other laboratory testing—a prolonged prothrombin time (PT), a prolonged partial thromboplastin time (PTT), decreased fibrinogen level, and elevated D-Dimer levels.¹⁷⁸ In VKDB, laboratory tests show a prolonged PT and a normal PTT.¹⁷⁹ When testing for the specific coagulation factors that are dependent on Vitamin K (factors II, VII, IX, and X), they are markedly decreased.¹⁸⁰ If findings and initial laboratory testing is concerning for VKDB, but vitamin K treatment has already been provided, measurement of proteins induced by vitamin K absence can confirm the diagnosis.¹⁸¹

The prevalence of ICH in people with acquired bleeding disorders is variable but very low. For example, the prevalence of ICH in patients with idiopathic ITP is <1%.¹⁸² In VKDB, administration of oral vitamin K prophylaxis reduces the incidence of late VKDB from 4.4–10.5/100,000 live births to 1.5–6.4/100,000 live births, and all patients with VKDB do not have ICH.¹⁸³

B. Symptoms of bleeding disorders

Although bleeding disorders, by definition, cause bleeding, the manifestations of bleeding disorders vary based upon location on the body, frequency, and severity. Some bleeding disorders, such as the mild platelet abnormalities and VWD, generally cause mild symptoms, such as mouth and/or nose bleeding or mild skin bruising.¹⁸⁴ Often, mild platelet abnormalities and VWD cause no symptoms at all.¹⁸⁵ More severe conditions, such as some types of hemophilia, often cause severe joint bleeding and may cause ICH.¹⁸⁶

¹⁷⁷ See Monagle & Andrew, *supra* note 147, at ____.

¹⁷⁸ *Id.* at ____.

¹⁷⁹ Martin J. Shearer, *Vitamin K Deficiency Bleeding (VKDB) in Early Infancy*, 23 BLOOD REVIEWS 49, 50 (2009).

¹⁸⁰ *Id.* at 53. In patients who have already received vitamin K as treatment or transfusion of plasma, measurement of proteins induced by vitamin K absence (PIVKA II) can confirm the diagnosis. *Id.*

¹⁸¹ *Id.* at 50-51.

¹⁸² Cindy Neunert et al., *The American Society of Hematology 2011 Evidence-Based Practice Guidelines for Immune Thrombocytopenia*, 117 BLOOD 4190, 4195 (2011).

¹⁸³ Alvin Zipursky, *Prevention of Vitamin K Deficiency Bleeding in Newborns*, 104 BRIT. J. HAEMATOLOGY 430, 432-33 (1999).

¹⁸⁴ Heather McKay et al., *Bleeding Risks Associated with Inheritance of the Quebec Platelet Disorder*, 104 BLOOD 159, 164 (2004).

¹⁸⁵ *Id.*

¹⁸⁶ P. Mishra et al. *Intracranial Haemorrhage in Patients with Congenital Haemostatic Defects*, 14 HAEMOPHILIA 952, 952-53 (2008); M. D. Nelson Jr. et al. *Prevalence and Incidence of Intracranial Haemorrhage in a Population*

When young children present with ICH and no history of trauma or a history of a minor trauma, one must consider a bleeding disorder as the underlying cause. ICH occurs more frequently in some bleeding disorders, very rarely in others, and either exceedingly rarely or not at all in other bleeding disorders.¹⁸⁷ As will be discussed below, the prevalence of each bleeding disorder and the prevalence of ICH within the population of people with that specific bleeding disorder may be used to identify a testing scheme to evaluate for bleeding disorders as a cause of ICH. However, prior to using the existing data to construct an evidence-based approach to evaluating for bleeding disorders in the setting of alleged AHT, it is necessary to first examine the data for validity and applicability.

C. Sources of data

Nearly all of the existing data regarding bleeding disorders and ICH have been culled from large databases, such as the Universal Data Collection (UDC) database project of the Centers for Disease Control.¹⁸⁸ The UDC was established in 1997 to monitor the safety of the blood supply in the United States and to track the incidence and consequences of joint complications in patients with bleeding disorders.¹⁸⁹ The UDC and other similar databases were not constructed to guide forensic evaluations regarding possible AHT and bleeding disorders. As a result, the details of the bleeding symptoms in the databases have not been collected in a fashion that would be preferable for forensic purposes.¹⁹⁰ For example, specifics such as trauma history, location of ICH (subarachnoid/subdural, etc.), and external evidence of trauma have not been collected. However, this does not eliminate the utility of the UDC and other databases in forensic consideration.

of Children with Haemophilia, 5 HAEMOPHILIA 306, 311 (1999); Rachelle Nuss et al., *Changes in the Occurrence of and Risk Factors for Hemophilia-Associated Intracranial Hemorrhage*, 68 AM. J. HEMATOLOGY 37, 40 (2001).

¹⁸⁷ See Carpenter et al., *supra* note 141, at ____.

¹⁸⁸ R. Kulkarni et al, *Sites of Initial Bleeding Episodes, Mode of Delivery and Age of Diagnosis in Babies with Haemophilia Diagnosed Before the Age of 2 Years: A Report from the Centers for Disease Control and Prevention's (CDC) Universal Data Collection (UDC) project*, 15 HAEMOPHILIA 1281 (2009).

¹⁸⁹ A.D. Metjian et al, *Bleeding Symptoms and Laboratory Correlation in Patients with Severe von Willebrand Disease*, 15 HAEMOPHILIA 918, 919 (2009).

¹⁹⁰ Ideally, the scientific literature would include studies that have systematically compared trauma histories, locations of bleeding, amount of bleeding, clinical presentations and outcomes, and the presence of retinal hemorrhages in children with and without bleeding disorders. However, given the rarity of most bleeding disorders, such studies would be extremely difficult and costly to conduct; as such, no such study exists.

The existing scientific literature generated from these hematology databases is useful in determining the prevalence of particular bleeding disorders in our population and the probability of a particular bleeding disorder to cause ICH in general. Even in the absence of large studies evaluating the forensic implications of bleeding disorders, the existing literature of case series and case reports is valuable.¹⁹¹ In the largest study examining non-accidental injury and bleeding disorders, Jackson et al. documented presentations of bleeding disorders over a ten-year period at a large pediatric center.¹⁹² After excluding patients diagnosed at birth with a bleeding disorder, 15.3% of all children with bleeding disorders presented in a manner that may be confused with abuse, including genital and buttock bruising, bruising in immobile infants, and ICH.¹⁹³ Five children in the study were involved with the legal or child protection system due to bleeding/bruising.¹⁹⁴ The authors concluded that bleeding disorders can present in a manner that is “clinically indistinguishable from abuse.”¹⁹⁵

However, of note, *only two* children over the study’s ten-year interval presented with ICH, both of whom were older than one year of age, had obvious evidence of impact (skull fractures), and VWD.¹⁹⁶ The authors were unable to determine if the findings in these cases were due to abuse or accidental impact.¹⁹⁷ There were *no cases of spontaneous ICH* (with no evidence of impact) in the study.¹⁹⁸ This suggests that spontaneous ICH due to a bleeding disorder is an extremely rare event.

D. Testing for and probability of bleeding disorders in the setting of ICH

Studies have evaluated the use of screening questions and family history in the detection of bleeding disorders.¹⁹⁹ Negative screens (i.e. the family or child has no history of easy

¹⁹¹ A. E. O’Hare & O. B. Eden, *Bleeding Disorders and Non-Accidental Injury*, 59 ARCHIVES OF DISEASE IN CHILDHOOD 860, 863 (1984); P.G. Scimeca et al., *Suspicion of Child Abuse Complicating the Diagnosis of Bleeding Disorders*, 13 J. PEDIATRIC HEMATOLOGY/ONCOLOGY 179, ____ (1996).

¹⁹² Jami Jackson et al., *Challenges in the Evaluation for Possible Abuse: Presentations of Congenital Bleeding Disorders in Childhood*, 36 CHILD ABUSE & NEGLECT 127, 131 (2012).

¹⁹³ *Id.* at 130.

¹⁹⁴ *Id.* at 131.

¹⁹⁵ *Id.*

¹⁹⁶ *Id.*

¹⁹⁷ *Id.*

¹⁹⁸ *Id.*

¹⁹⁹ See Francesco Rodeghiero et al., *The Discriminant Power of Bleeding History for the Diagnosis of Type 1 von Willebrand Disease: An International, Multicenter Study*, 3 J. THROMBOSIS & HAEMOSTASIS 2619, 2619-20 (2005).

bleeding/bruising) are not effective ways of ruling out a bleeding disorder.²⁰⁰ Similarly, statements such as “my child bruises easily” or a family history of “easy bruising” do not rule out abuse as a cause of bleeding in a child.²⁰¹ Thus, when clinicians consider the potential need for testing for bleeding disorders, in the absence of a known, named bleeding disorder in the child or family, the family history of bleeding/bruising is of limited use.

When ordering laboratory tests for clinical or forensic reasons, clinicians must ask, “What is the potential for a positive test result?” and “How is the result of this test going to change my forensic impression or patient management?” If the potential for a positive test result is microscopically small or if a positive test result does not change the clinical impression/diagnosis, there is very little value in sending the test. For instance, the prevalence of Factor 2 (prothrombin) deficiency (1 per 1 million people) and frequency of ICH within the population of people with Factor 2 (prothrombin) deficiency (11%) make testing for Factor 2 (prothrombin) deficiency of extremely low value on cases of suspected AHT.²⁰²

Because there is a remote potential for a bleeding disorder presenting as ICH, clinicians often consider evaluating for such in cases of possible AHT.²⁰³ However, when clinically assessing a particular child, the entire set of clinical and historical findings must be considered together.²⁰⁴ If a child has other findings that are highly suggestive of violent trauma (e.g., fractures) or other findings that are unrelated to bleeding disorders, it is reasonable to exclude testing for bleeding disorders.²⁰⁵ An evaluation for bleeding disorders is generally performed if a child has ICH with no readily apparent explanation or with no other evidence strongly suggesting abuse (e.g., unexplained fractures, witnessed abuse, patterned bruising) as this may rarely be the presenting manifestation of a bleeding disorder.²⁰⁶

A basic tenet of practicing evidenced-based medicine is that actual evidence rather than hypotheses can be used to guide clinical decision-making.²⁰⁷ Hypothetical considerations without proven cause-effect linkage to ICH, such as vaccines²⁰⁸ and “choking episodes,”²⁰⁹

²⁰⁰ *Id.* at 2624.

²⁰¹ *See* Jackson, *supra* note 167, at 127.

²⁰² *See* Carpenter, *supra* note 141, at ____; Jackson, *supra* note 167, at 128.

²⁰³ *See* Jackson, *supra* note 167, at 127-28.

²⁰⁴ *Id.*

²⁰⁵ *Id.*

²⁰⁶ *See* Carpenter, *supra* note 141, at ____; Jackson, *supra* note 167, at 127-28.

²⁰⁷ *See* CEBM, *supra* note 26.

²⁰⁸ *See* Gardner, *supra* note 140, at 663.

cannot be considered evidence-based medicine. Thus, testing for histamine levels or vitamin C levels based on a hypothesis that vaccines induce ICH by altering levels of those factors is not grounded in scientific, evidence-based practice.²¹⁰ Such hypotheses should be tested by rigorous prospective research prior to being offered as an explanation for ICH in a legal setting.

When deciding on which tests to order to evaluate for a bleeding disorder as the cause of ICH in a young child, clinicians can access the existing data on the prevalence of specific bleeding disorders and the prevalence of ICH due to those specific bleeding disorders. If the prevalence of a condition and the frequency of a particular presentation of that condition are known, a physician can construct the probability of that specific condition (bleeding disorder) resulting in the specific presentation (ICH):

$$\text{PTP(B)} = \text{P(A)} \times \text{P(B|A)}^{211}$$

For example, severe VWD is extremely rare, occurring at an upper-limit estimated population prevalence of 1 per 300,000 people.²¹² Up to 4% of people with severe VWD initially present with a “head bleed,” including both ICH and extracranial (scalp or facial) bleeding.²¹³ Thus, the estimated probability that a person will get an ICH due to severe VWD is:

$$\begin{aligned} &(\text{Prevalence of severe VWD}) \times (\text{Prevalence of ICH in severe VWD}) \\ &(1/300,000) \times (0.04) = 1/7.5 \text{ million} \end{aligned}$$

The calculated probability, in this instance, is actually an *overestimate* of severe VWD causing subdural hemorrhage (SDH) because the calculated probability pertains to not only all ICH (SDH, subarachnoid hemorrhages, and epidural hematomas and parenchymal²¹⁴ bleeding) but scalp and facial bleeding as well. Additionally, spontaneous SDH (i.e., those not resulting

²⁰⁹ Patrick D. Barnes et al., *Infant Acute Life-Threatening Event-Dysphagic Choking Versus Nonaccidental Trauma*, 17 SEMINARS IN PEDIATRIC NEUROLOGY 7, 9-10 (2010).

²¹⁰ See Clemetson, *supra* note 140, at 535.

²¹¹ Where A = a specific bleeding disorder; B = ICH due to bleeding disorder A; P = Probability/Prevalence, and PTP = Pre-test Probability

²¹² See Metjain, *supra* note 164, at 918, 922.

²¹³ *Id.* at 921.

²¹⁴ “Parenchymal” bleeding refers to bleeding within the brain matter itself, not outside it like SDH, subarachnoid hemorrhage or epidural hemorrhage.

from trauma) is a subset of all SDHs. Thus, it is reasonable for a clinician to conclude that the chances a young child will suffer SDH (either spontaneously or as a result of trauma) from a previously undiagnosed, severe VWD are exceedingly small.

Similar probabilities can be calculated for any bleeding disorder in which: 1) the prevalence of the condition is known and 2) the prevalence of ICH within the condition is known (*See Table 1 below*).

Table 1: Probabilities for Congenital Coagulopathies to Cause ICH^{a,b}

Condition	Prevalence of Condition (Upper Limits)	Prevalence of ICH (Upper Limits)	Probability
VWD	1 per 1000	Extremely rare	<i>Low</i>
Factor II deficiency	1 per 1 million	11%	<i>1 per 10 million</i>
Factor V deficiency	1 per 1 million	8% of homozygotes	<i>1 per 10 million homozygotes</i>
Combined factors V and VIII deficiencies	1 per 1 million	2%	<i>1 per 50 million</i>
Factor VII deficiency	1 per 300,000	4–6.5%	<i>1 per 5 million</i>
Factor VIII deficiency	1 per 5000 males	5–12%	<i>1 per 50,000 males</i>
Factor IX deficiency	1 per 20,000 males	5–12%	<i>1 per 200,000 males</i>
Factor X deficiency	1 per 1 million	21%	<i>1 per 5 million</i>
Factor XI deficiency	1 per 100,000	Extremely rare	<i>Low</i>
Factor XIII deficiency	1 per 2 million	33%	<i>1 per 6 million</i>
Alpha-2 antiplasmin deficiency	40 cases reported	Not reported	<i>Low</i>
Plasminogen activator inhibitor-1 deficiency (PAI-1)	Extremely rare	Common	<i>Low</i>
Afibrinogenemia	1 per 500 000	10%	<i>1 per 5 million</i>

Dysfibrinogenemia	1 per 1 million	Single case reported	<i>Low</i>
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^a The probability of having a specific bleeding disorder increases in the setting of a family history of that specific-named bleeding disorder or if the patient is from an ethnicity in which a specific bleeding disorder is more common (e.g., Ashkenazi Jewish people and factor XI deficiency).

^b “Probability” indicates the probability that an individual in the general population would have the following specific coagulopathy causing an intracranial hemorrhage.

Used with permission from ____ Carpenter et al., *Technical Report: Evaluating for Suspected Child Abuse: Conditions that Predispose to Bleeding*, PEDIATRICS (In Press).

For instance, the two most common severe congenital bleeding disorders, Factor 8 and Factor 9 deficiencies (two forms of hemophilia), have probabilities for ICH of 1 per 50,000 males and 1 per 200,000 males, respectively.²¹⁵ The probability of Factor 13 deficiency causing an ICH is 1 per 6 million, largely due to the rarity of Factor 13 deficiency.²¹⁶ This means that, in the population in general, a single person’s risk of having an ICH due to Factor 13 deficiency is 1 in 6 million.

Ordering tests to evaluate for every bleeding disorder is generally impractical due to the statistical implausibility of many of the bleeding disorders causing ICH. In fact, many conditions have a probability of causing ICH so low as to preclude calculation. However, these probabilities can be used to identify the need for tests to be ordered if a child’s findings may reasonably be caused by a bleeding disorder. Thus, physicians may order tests with higher (relatively speaking) probabilities, such as evaluating for conditions that have a probability higher than or equal to 1 in 5 million, for instance. *If negative test results are obtained, the post-test probability of one of the tested bleeding disorders is essentially zero.*

E. Special considerations

Two bleeding disorders have particular potential to create confusion or pose a diagnostic challenge in the evaluation of possible AHT—von Willebrand disease and mild platelet disorders.

²¹⁵ See Carpenter, *supra* note 141, at _____. Due to the method by which these forms of hemophilia are inherited, severe hemophilia is much more common in males than females.

²¹⁶ See Table 1.

1. *Von Willebrand Disease (VWD)*

VWD is the most common congenital bleeding disorder.²¹⁷ It most commonly presents with mild to moderate bleeding from the nose or mouth, bruising, or heavy bleeding during a woman's menstrual period.²¹⁸ It is generally classified in terms of von Willebrand factor (VWF) levels and the type of functional defect affecting the VWF protein.²¹⁹ Type 1 disease results from an absolute decrease in the VWF protein and is the most common.²²⁰ Type 3 is characterized by nearly absent levels of VWF as well as low factor 8 and is the most severe version of VWD.²²¹ There are also a number of qualitative abnormalities resulting in variable bleeding manifestations (types 2a, 2b, 2m, and 2n).²²² Testing for VWD can be complicated and often requires consultation with a pediatric hematologist.²²³

The current prevalence of VWD is difficult to determine²²⁴ as recent consensus changes have resulted in more specific diagnostic criteria.²²⁵ Per the National Heart, Lung, and Blood Institute, the most recent criteria for diagnosis requires VWF levels <30% (normal range 50–150%), resulting in fewer individuals with levels below the normal range meeting diagnostic criteria.²²⁶ Current estimates indicate that low VWF levels may occur in up to 1% of the US population.²²⁷ However, since many persons with VWD do not manifest symptoms, the

²¹⁷ W.L. Nichols et al., *Von Willebrand Disease (VWD): Evidence-Based Diagnosis and Management Guidelines, the National Heart, Lung, and Blood Institute (NHLBI) Expert Panel Report (USA)*, 14 HAEMOPHILIA 171, 171 (2008).

²¹⁸ *Id.* at 197.

²¹⁹ *Id.* at 172.

²²⁰ *Id.* at 178, 179.

²²¹ *Id.* at 181.

²²² *Id.* at 179-80.

²²³ *The Diagnosis, Evaluation, and Management of von Willebrand Disease*, NIH publication No. 08-5832, U.S. DEP'T OF HEALTH AND HUMAN SERV., NATIONAL INST. OF HEALTH, NATIONAL HEART LUNG AND BLOOD INST., at 2, 32, 41, 49-50 (2007) (stating that repeat testing for VWD may be required because VWF levels may be artificially elevated in times of critical illness). Testing for VWD includes von Willebrand factor (VWF) levels, VWF activity, and Factor 8 levels.

²²⁴ *Id.* at 31-32. Bleeding symptoms of VWD are generally mild; therefore, it is reasonably assumed by clinicians that there are people with low VWF levels who have not come to medical attention.

²²⁵ *Id.*

²²⁶ *Id.* at 16. Individuals with bleeding symptoms and VWF levels between 30-50% currently pose a diagnostic dilemma for clinicians.

²²⁷ *Id.* at 1.

prevalence of *symptomatic* persons with VWD is currently best estimated at 23–110 per million or 0.0023–0.01% of the US population.²²⁸

*Additionally, and more importantly, ICH as the presenting finding of severe VWD is extremely rare (upper limit of probability of 1 per 7.5 million people).*²²⁹ Mild VWD is much more common than severe VWD, but the prevalence of ICH within people with mild VWD is unknown.²³⁰ Because low VWF levels are relatively common, it is certain that testing for VWD will be positive in a small percentage of children with findings concerning for AHT.

*In nearly all cases, where VWD is mild, it often does not cause any symptoms until a hemostatic challenge, like the removal of teeth.*²³¹ A review of the peer-reviewed literature in humans on ICH due to VWD reveals four published cases of spontaneous ICH in adults.²³² None of these cases involved subdural hemorrhage.²³³ The peer-reviewed medical literature evaluating mild trauma and VWD in humans contains only a few published case reports and case series of VWD complicating mild or more severe trauma in mobile children or adults.²³⁴ Only one of these cases involved subdural hemorrhage—that case being a four-year-old child who suffered an impact to the head.²³⁵ *There is currently no scientific data to support the hypothesis that VWD is a cause of spontaneous ICH in young, immobile children. And the best current scientific literature supports the conclusion that VWD may very rarely contribute to bleeding complications in mild head trauma.*

The clinician is posed with a diagnostic challenge when the historical and clinical findings are consistent with AHT, but laboratory testing shows low VWF levels. Some

²²⁸ J.E. Sadler et al., *Impact, Diagnosis and Treatment of von Willebrand Disease*, 84 J. THROMBOSIS & HAEMOSTASIS, 160, 164 (2000).

²²⁹ See W.S. Almaani et al., *Spontaneous Intracranial Hemorrhage Secondary to von Willebrand Disease*, 26 SURGICAL NEUROLOGY 457, 458 (1986); Kazuo Mizoi et al., *Intracranial Hemorrhage Secondary to von Willebrand's Disease and Trauma*, 22 SURGICAL NEUROLOGY 495, 498 (1984).

²³⁰ *Id.*

²³¹ See Nichols, *supra* note 184, at 186-87.

²³² See Almaani, *supra* note 195, at 457.

²³³ *Id.* at 458.

²³⁴ See Mizoi, *supra* note 195, at 495-98; O. Ziv & M. V. Ragni, *Bleeding Manifestations in Males with von Willebrand Disease*, 10 HAEMOPHILIA 162, 162-168 (2004); T. Delangre et al., *Hematome Intracerebral du a Une Forme Attenuée de Maladie de von Willebrand*, 15 LA PRESSE MEDICALE 1240, 1240-1241 (1986); Robert Diecidue et al., *Post-Traumatic Hemorrhage in a Patient with Previously Undiagnosed von Willebrand's Disease*, 58 J. ORAL MAXILLOFACIAL SURGERY 332, 332-336 (2000); Kristine Slam et al., *Common Bleeding Disorders: A Potential Catastrophe for the Trauma Victim. Therapeutic Recommendations for the Treatment of von Willebrand's Disease*, 64 J. TRAUMA 1373, 1373-1375 (2008).

²³⁵ See Mizoi, *supra* note 195, at 495.

individuals have concluded that laboratory tests consistent with VWD essentially eliminate the consideration of AHT.²³⁶ Others have been more balanced, stating that, “The significance of von Willebrand disease as a possible contributory factor in infants with subdural and retinal hemorrhages should be further addressed.”²³⁷ The notion that laboratory testing consistent with VWD “rules out” AHT as a diagnosis is not only irrational (as the presence of VWD does not protect a child from AHT), but is also unsupported by the scientific literature.²³⁸

2. Mild platelet disorders

Congenital platelet disorders can result in fewer platelets, abnormal function of platelets, or a combination of the two.²³⁹ Mild congenital platelet disorders include Quebec platelet disorder, the MYH9 related disorders, Scott syndrome, Hermansky-Pudlak syndrome, Chediak-Higashi syndrome, and Wiskott-Aldrich syndrome.²⁴⁰ Specific testing for platelet function is required to detect these disorders.²⁴¹ *Most bleeding with these disorders is mild and manifests as excessive bruising or menorrhagia (heavy menstrual periods).*²⁴²

The exact prevalence of mild platelet disorders is unknown.²⁴³ *The probability of mild platelet disorders causing ICH is also unknown but is likely very low given the typical clinical manifestations.*²⁴⁴ Much like VWD, if specific testing is performed in children with suspected abusive ICH, it is likely that a small number of children will have laboratory results indicative of

²³⁶See Martha E. Laposata & Michael Laposata, *Children with Signs of Abuse. When is it Not Child Abuse?* 123 AM. J. CLINICAL PATHOLOGY (Supp. 1) S119 (2005).

²³⁷Arne Stray-Pedersen et al., *An Infant with Subdural Hematoma and Retinal Hemorrhages: Does von Willebrand Disease Explain the Findings?*, 7 FORENSIC SCI. MED. PATHOLOGY 37, 41 (2011).

²³⁸See Kazuo Mizoi et al., *supra* note __; O. Ziv & M. V. Ragni, T. Delangre et al., Kristine Slam et al., *supra* note 200. Additionally, the child with documented mild VWD and accidental head trauma was a 4-year-old who suffered an accidental fall with a resulting large subdural hematoma that required surgical drainage. This clinical picture is significantly different from the vast majority of child victims of AHT where the children are: less than 1-year-old, immobile, often with no external signs of impact to the head, and with thin, bilateral convexity subdural hematomas.

²³⁹Catherine P. M. Hayward et al., *Congenital Platelet Disorders: Overview of Their Mechanisms, Diagnostic Evaluation and Treatment*, 12 HAEMOPHILIA (Supp. 3) 128, 129 (2006).

²⁴⁰*Id.* at 132; Otobia Dimson et al., *Hermansky-Pudlak Syndrome*, 16 PEDIATRIC DERMATOLOGY 475, 475-77 (1999).

²⁴¹Emmanuel J. Favaloro, *Clinical Utility of the PFA-100*, 34 SEMINARS THROMBOSIS HEMOSTASIS 709, 709-710 (2008); Hayward et al., *supra* note 205, at 133. The more specific platelet testing would assess platelet aggregation and secretion. Occasionally, electron microscopic examination or genetic testing is necessary to confirm the diagnosis.

²⁴²See Hayward et al., *supra* note 205, at 132.

²⁴³*Id.* at 130.

²⁴⁴*Id.*

a mild platelet disorder.²⁴⁵ In these cases, laboratory results do not rule out AHT, as there are currently no scientific data to support the hypothesis that mild platelet disorders have caused a spontaneous ICH.²⁴⁶

Rare *severe* congenital platelet disorders, such as Bersonard-Soulier syndrome (BSS) and Glanzmann thrombasthenia (GT) are known causes of ICH.²⁴⁷ In both of these disorders, significant mucocutaneous bleeding and ICH have been reported, although ICH is rare, occurring in only 0.3–2% of patients with GT and even less in those with BSS.²⁴⁸ Screening for these disorders may be accomplished using the Platelet Function Analyzer test (PFA-100).²⁴⁹

F. Conclusion

The evaluation of level of evidence for the probability that a bleeding disorder caused an ICH is a “symptom prevalence” question. The most recent Oxford Centre for Evidence-Based Medicine levels of evidence ratings²⁵⁰ do not address these types of questions. However, the 2009 version of the Oxford Centre for Evidence-Based Medicine does address “symptom prevalence” questions and is an appropriate evaluation tool.²⁵¹ There are a large number of studies evaluating the potential for ICH in patients with specific bleeding disorders with variable levels of evidence.

*Any studies based on prospective registries, such as the Universal Data Collection of the Centers for Disease Control and Prevention and the North American Rare Bleeding Disorder Registry, qualify as “1b” according to the 2009 Oxford levels.*²⁵² Most other studies on the subject are best classified as 1c and 2b. Any individual claiming that an ICH might be the result of a “bleeding disorder” should specify which bleeding disorder is of concern, the general

²⁴⁵ Laposata, *supra* note 202, at S120.

²⁴⁶ See Kzuo Mizoi et al., *supra* note __; O. Ziv & M.V. Ragni, T. Delangre et al., KristineSlam et al., *supra* note 200; Joseph Broderick et al., *Stroke*, 38 AM. HEART ASS’N J.2001-23 (2007), available at <http://stroke.ahajournals.org/content/38/6/2001.full>.

²⁴⁷ Giovanni Di Minno et al., *Glanzmann’s Thrombasthenia (Defective Platelet Integrin αII_b - β_3): Proposals for Management Between Evidence and Open Issues*, 102 THROMBOSIS HEMATOSIS 1157, 1158 (2009); Jayanthi Alamelu & Ri Liesner, *Modern Management of Severe Platelet Function Disorders*, 149 BRIT. J. HAEMATOLOGY 813, 813 (2010).

²⁴⁸ Di Minno et al., *supra* note 211, at 1157-1158; Amaelu & Liesner, *supra* note 211 at 813.

²⁴⁹ See Hayward et al., *supra* note 205, at 133; Favaloro, *supra* note 206, at 709.

²⁵⁰ See CEBM website, *supra* note 26.

²⁵¹ See Bob Phillips et al., *Oxford Centre for Evidence-Based Medicine – Levels of Evidence (March 2009)*, CEBM, <http://www.cebm.net/index.aspx?o=1025> (last edited Jan. 29, 2013).

²⁵² See, e.g., Acharya et al., *supra* note 142.

prevalence of that bleeding disorder in the population, and the probability that that specific bleeding disorder *causes* ICH.

Biomechanics

Classical mechanics began with the work of Isaac Newton in the late 1600s.²⁵³ Beginning with a few simple equations, engineers can predict how many objects will respond to various forces.²⁵⁴ One branch of mechanics, biomechanics, concerns itself with “the scientific study of mechanics in biological systems.”²⁵⁵

In contrast to the observational, clinical studies noted in the accidents section above, biomechanics is fundamentally an experimental discipline. Biomechanical engineers, like all responsible scientists, are unwilling to injure living children in the course of an experiment. Instead, engineers employ a number of approximations, ranging from animals to constructed “crash test dummies” to finite element analysis (FEA). Each of these techniques has some value, and none is perfect. The best scientific insight results from a careful consideration of the strengths and limitations of all available information.

Traumatic brain injury can result from either inertial (a rapid head acceleration-deceleration that produces injurious brain deformation) or contact (where impact produces local brain deformations) mechanisms.²⁵⁶ Although rapid brain acceleration can proceed in either linear or rotational directions, it is actually high angular accelerations and velocity that are often correlated with intracranial hemorrhage and severe brain injury.²⁵⁷

Traditionally, biomechanical analysis of head injury in infants and children assumed that infants and young children responded like small adults.²⁵⁸ Using an engineering approach called “dimensional analysis,” it was assumed that critical inertial loading conditions for severe brain injuries (such as SDHs or diffuse axonal injury) could be scaled from adults to infants based

²⁵³ISAAC NEWTON, PHILOSOPHIAE NATURALIS PRINCIPIA MATHEMATICA (London, S. Pepys, Reg. Soc. Praeses 1686), *available at* <http://www.gutenberg.org/files/28233/28233-pdf>.

²⁵⁴*See, e.g.,* BENJAMIN CROWELL, MECHANICS 14 (2012), *available at* <http://www.lightandmatter.com/me.pdf>.

²⁵⁵THE AMERICAN HERITAGE SCIENCE DICTIONARY (Joseph P. Pickett et al. eds., 2005).

²⁵⁶Susan Margulies & Brittany Coats, *Biomechanics of Head Trauma in Infants and Young Children*, in CHILD ABUSE AND NEGLECT: DIAGNOSIS, TREATMENT AND EVIDENCE 359, 359 (Carole Jenny ed., 2011).

²⁵⁷*Id.* at 359; *See also* Ramesh Raghupathi & Susan S. Margulies, *Traumatic Axonal Injury After Closed Head Injury in the Neonatal Pig*, 19 J. NEUROTRAUMA 843, (2002).

²⁵⁸Margulies & Coats, *supra* note 220, at 359.

solely upon brain mass.²⁵⁹ However, recent biomechanical studies²⁶⁰ have shown what pediatricians have long argued—that *children are not small adults*. Multiple differences—in tissue composition, brain and skull properties, and brain vulnerability—between adults and children have prompted scientists to interpret biomechanical studies that utilize scaling approaches with caution.²⁶¹

The biomechanics of AHT/SBS is a comprehensive and complex topic. A few biomechanical questions are frequently encountered in AHT/SBS. We will summarize the scientific literature pertinent to those questions. For a more comprehensive discussion of the topic, we refer the reader to additional literature.²⁶²

Question 1: Does the biomechanical literature suggest that shaking alone cannot cause SDH or serious brain injury in children?

As Narang mentioned in his first article, when traumatic, SDHs are caused by rupture of the bridging veins in the brain.²⁶³ Early biomechanical work by Gennarelli and Thibault in primates revealed a strong association of SDH with elongation of the bridging veins beyond their strain tolerances when the primate brains moved relative to their skulls during sudden acceleration-deceleration events.²⁶⁴ In 1987, Duhaime et al. published a biomechanical study of shaken baby syndrome in which the authors shook a constructed model infant.²⁶⁵ Duhaime suggested that impact was necessary to cause SDH because shaking alone achieved maximum velocities and accelerations that were well below the thresholds for SDH (*that were scaled from adult primates*) and impact exceeded those thresholds.²⁶⁶ This study created a tide of misplaced sentiment that shaking alone could not cause significant injury in infants and children.²⁶⁷

²⁵⁹ *Id.*

²⁶⁰ Nicole G. Ibrahim et al., *Physiological and Pathological Responses to Head Rotations in Toddler Piglets*, 27 J. NEUROTRAUMA 1021, 1021 (2010).

²⁶¹ See Margulies & Coats, *supra* note 220, at 359.

²⁶² See CAROLE JENNY, CHILD ABUSE AND NEGLECT: DIAGNOSIS, TREATMENT AND EVIDENCE (Elsevier Saunders 2011).

²⁶³ Narang, *supra* note 10, at 542.

²⁶⁴ See Margulies & Coats, *supra* note 220, at 359, 362 (citing to works published earlier by Thomas A. Gennarelli and Lawrence E. Thibault, *supra* notes 13, 14, 15, and 18).

²⁶⁵ Ann-Christine Duhaime, et al., *The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study*, 66 J. NEUROSURGERY 409, 409 (1987).

²⁶⁶ C.Z. Cory & M.D. Jones, *Can Shaking Alone Cause Fatal Brain Injury? A Biomechanical Assessment of the Duhaime Shaken Baby Syndrome Model*, 43 MED. SCI. & LAW 317, 317 (2003).

²⁶⁷ *Id.*

Scientific critique of the Duhaime study has highlighted the importance of biofidelity in doll models. Cory and Jones²⁶⁸ found that making just minimal adjustments to Duhaime's model (such as altering the center of gravity in the head) created a model in which manual shaking did exceed injury thresholds in 8 out of 10 trials.²⁶⁹ Wolfson et al.'s FEM study determined that slight modifications of the stiffness and hinge used in the model's neck dramatically altered the rotational accelerations and velocities achieved.²⁷⁰ These studies underscore the principle that even the slightest variation in the experimental model can result in aberrant data.

Further studies have shown that other factors not considered in Duhaime's study also affect the likelihood of injury. Eucker showed that the direction of head rotation (back and forth versus side to side.) affects likelihood of injury.²⁷¹ Prins et al. suggested that both adult and infant brains are increasingly susceptible to repetitive injury (i.e., implying a cumulative effect of injury).²⁷² Finally, Kochanek and colleagues have mounted evidence that biochemical and metabolic responses to brain injury are significantly different in the young infant compared to the older child or adult.²⁷³

In 2003, Prange et al. performed an updated version of the Duhaime's study.²⁷⁴ Like Duhaime's prior study, Prange et al. compared biomechanical forces achieved from shaking and shaking with impact (which ended with forceful impact onto a rigid or padded surface but without throwing the model).²⁷⁵ The authors concluded that "[v]igorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted

²⁶⁸ *Id.* at 317..

²⁶⁹ Cory and Jones also proposed different injury thresholds, which were lower than in the Duhaime study. The requirement of the experimenter to select a number, or threshold, at which injury is presumed to have occurred, is a significant weakness of a constructed model study. *Id.* at 320.

²⁷⁰ D.R. Wolfson, et al., *Rigid-Body Modelling of Shaken Baby Syndrome*, 219 PROC. INST. MECHANICAL ENGINEERING (Part H: J. Engineering Med.) 63, 66 (2005).

²⁷¹ Stephanie A. Eucker, et al., *Physiological and Histopathological Responses Following Closed Rotational Head Injury Depend on Direction of Head Motion*, 227 EXPERIMENTAL NEUROLOGY 79, 85 (2011).

²⁷² Mayumi L. Prins et al., *Repeated Mild Traumatic Brain Injury: Mechanisms of Cerebral Vulnerability*, 30 J. NEUROTRAUMA 30, 35 (2013).

²⁷³ See Randy A. Ruppel et al., *Critical Mechanisms of Secondary Damage After Inflicted Head Injury in Infants and Children*, 13 NEUROSURGERY CLIN. N. AM. 169, 169-82 (2002); Patrick M. Kochanek et al., *Biomedical, Cellular, and Molecular Mechanisms in the Evolution of Secondary Damage After Severe Traumatic Brain Injury in Infants and Children: Lessons Learned from the Bedside*, 1 PEDIATRIC CRITICAL CARE MED. 4, 4-19 (2000).

²⁷⁴ Michael T. Prange et al., *Anthropomorphic Simulations of Falls, Shakes and Inflicted Impacts on Infants*, 99 J. NEUROSURGERY 143, 143-50 (2003).

²⁷⁵ *Id.*

impacts produced responses that were significantly higher than even a 1.5m fall onto concrete.”²⁷⁶

Contrasting Duhaime’s and Prange’s findings, a host of biomechanical studies have yielded different conclusions. In a FEM study by Roth et al., the authors questioned whether angular acceleration—the value for which Duhaime and Prange compared injury thresholds—could accurately predict injury.²⁷⁷ Roth et al. recreated the injury events in the Prange study using a computational model.²⁷⁸ In spite of dramatic differences in rotational acceleration, shaking and impact created similar strain on the bridging veins.²⁷⁹ Because shaking and impact cause similar strains on the bridging veins, it is not unreasonable to expect similar injuries to result.²⁸⁰

A valuable FEM study by Morison examined the protective effect of cerebrospinal fluid (CSF) on intracranial injury.²⁸¹ The CSF is an important but difficult to model structure that is notably absent from Duhaime’s and Prange’s models.²⁸² Morison demonstrated that the CSF dampens brain acceleration to 0.13–1.00% of translational skull acceleration *but does not protect the brain from rotational acceleration*.²⁸³ Duhaime’s and Prange’s studies did not incorporate or study Morison’s assertion that cerebrospinal fluid protects the brain against a short fall but not from shaking.

Finally, Finnie et al. studied shaking in actual living animals.²⁸⁴ The authors grasped 7–10-day-old lambs under the axilla and vigorously shook them.²⁸⁵ The lamb model was selected principally because it has a relatively large gyrencephalic brain, large head, and weak neck

²⁷⁶*Id.* at 143.

²⁷⁷Sébastien Roth et al., *Finite Element Analysis of Impact and Shaking Inflicted to a Child*, 121 INT’L J. LEGAL MED. 223, 223-28 (2007).

²⁷⁸*Id.* at 224.

²⁷⁹*Id.* at 227; *see also* H. Maxeiner, *Detection of Ruptured Cerebral Bridging Veins at Autopsy*, 89 FORENSIC SCI. INT’L 103, 103-10 (1997) (Injury to the bridging veins is believed to be the cause of subdural hematomas in AHT).

²⁸⁰Limitations of the data in this study include the fact that the authors of the studies did not provide properties for vessels, and modeled the CSF as a solid material. Additionally, they modeled the vein as an elastic spring, which is not rate dependent.

²⁸¹Christopher Neil Morison, *The Dynamics of Shaken Baby Syndrome* (June 2002) (unpublished Ph.D. dissertation, University of Birmingham) (on file with the University of Birmingham Research Archive).

²⁸²*Id.* at 61.

²⁸³*Id.* at 82.

²⁸⁴John W. Finnie et al., *Diffuse Neuronal Perikaryal Amyloid Precursor Protein Immunoreactivity in an Ovine Model of Non-Accidental Head Injury (the Shaken Baby Syndrome)*, 17 J. CLINICAL NEUROSCIENCE 237(2010).

²⁸⁵*Id.*

muscles resembling a human infant.²⁸⁶ No lamb suffered impact to the head.²⁸⁷ Each lamb was shaken 10 times for 30 seconds.²⁸⁸ This methodology was based upon perpetrator confessions that indicate that repeated and violent shaking is common in AHT.²⁸⁹ Two of the seven lambs shaken in this manner suffered small SDHs,²⁹⁰ and two lambs had minor retinal hemorrhages.²⁹¹ Shaken lambs showed significantly more damage on microscopic examination compared to lambs that were not shaken.²⁹²

To honestly answer the question whether shaking alone can cause SDH and/or severe brain injury, the answer must assess all of the studies mentioned above, not just the Duhaime study of 1987. As discussed above, numerous authors have identified variables not considered by Duhaime that are important determinants of injury. Perhaps the most pointed criticism of these anthropometric model studies for determining injuries from shaking comes from the Prange paper itself:

These injury projections should be interpreted with caution, because differences in species, age, material properties, geometry, and direction make scaling experimental angular acceleration and velocity measurements to infants problematic *when based on differences in brain mass alone*. To avoid the limitations of using scaled loads from animal and cadaver experiments to investigate real life events, case studies of minor falls in infants were also used to examine injuries that occur as a result of falling from different heights. Unfortunately, these falls are rarely witnessed, load measurements of the event are lacking, contact surface information is rarely given, and the population studied generally includes a broad age range, rather than just newborns.²⁹³

In conclusion, in pediatric head injury, it must be remembered that *there are no human data on load tolerances*²⁹⁴ causing SDHs. What has been presented above is that the biomechanical literature does not offer a definitive “yes” or “no” answer to the widely debated question of whether shaking alone can cause SDHs. Some literature demonstrates it can occur,

²⁸⁶*Id.*

²⁸⁷*Id.* at 238.

²⁸⁸*Id.*

²⁸⁹Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 PEDIATRICS 546, 546-55 (2010).

²⁹⁰Finnie, *supra* note 242, at 238.

²⁹¹*Id.*

²⁹²*Id.*

²⁹³Prange, *supra* note 235, at 149 (emphasis added).

²⁹⁴“Load tolerance” means the magnitude of applied force necessary to cause injury to a biological tissues.

while other literature disputes it. It is clear, however, that continued assertion of the principle—that biomechanics clearly demonstrates that SDHs and/or serious brain injury cannot result from shaking—is disingenuous and scientifically irresponsible.

Question 2: Does biomechanics show that injurious shaking would necessarily cause catastrophic neck injury?

A 2005 paper by Bandak is widely cited as additional biomechanical evidence supporting the proposition that shaking alone cannot cause the injuries noted in AHT.²⁹⁵ In that paper, Bandak proposed that shaking sufficient to cause brain injury would necessitate devastating injuries to the cervical spine.²⁹⁶ Bandak's paper is purely analytic; meaning, rather than conducting original experiments, the purpose of the study was to reinterpret previously published data.²⁹⁷ Reinterpretation of published data is a valid study design and is the design of several of the studies discussed previously.

Bandak relied upon Jenny's²⁹⁸ reports of rotational acceleration and Duhaime's²⁹⁹ report of rotational velocities from adults shaking a model infant.³⁰⁰ Using these data, Bandak computed the amount of force experienced by the neck during these shaking events.³⁰¹ Bandak's computed forces greatly exceeded prior estimates of neck tensile strength in several animals.³⁰² Because children with AHT are frequently not noted to have severe neck injuries, Bandak **concluded** that a re-evaluation of AHT is needed.³⁰³

This work has suffered much scientific criticism. Three of the four studies of neck tensile strength³⁰⁴ do not involve humans and were presented with an explicit condition that they were preliminary and not to be used as references.³⁰⁵ The remaining article³⁰⁶ studied static rather

²⁹⁵Faris A. Bandak, *Shaken Baby Syndrome: A Biomechanical Analysis of Injury Mechanisms*, 151 FORENSIC SCI. INT'L 71, 71-79 (2005).

²⁹⁶*Id.* at 78.

²⁹⁷*Id.* at 71.

²⁹⁸C. Jenny et al., *Injury biomechanics research*, 30TH INTERNATIONAL WORKSHOP 129-143 (2002).

²⁹⁹Duhaime, *supra* note 228.

³⁰⁰Bandak, *supra* note 251, at 76.

³⁰¹*Id.* at 76-78.

³⁰²*Id.* at 77. See also J. Matthews Duncan, *Laboratory Note: On the Tensile Strength of the Fresh Adult Fetus*, 2 BR. MED. J. 763, 763-64 (1874); Randal P. Ching et al., *Tensile Mechanics of the Developing Cervical Spine*, 45 STAPP CAR CRASH 329, 329-36 (2001); R. Mayer et al., *Pediatric Tensile Neck Strength Characteristics Using a Caprine Model*, INJURY BIOMECHANICS RESEARCH, PROCEEDINGS OF THE 27TH INTERNATIONAL WORKSHOP ON HUMAN SUBJECTS BIOMECHANICS 87-92 (1999).

³⁰³Bandak, *supra* note 251, at 79.

³⁰⁴See Randal P. Ching, *supra* note _____. R. Mayer et al., *supra* note ____.

³⁰⁵N. Rangarajan & T. Shams, *Letter to the Editor*, 164 FORENSIC SCI. INT'L, 280, 281 (2006).

than dynamic loads imposed by a shaking baby.³⁰⁷ Other authors noted that neck injury is actually not as rare in AHT as Bandak asserted.³⁰⁸

The most serious criticism, however, is that Bandak's computations are simply incorrect. Stated simply, Bandak's math is wrong. This is especially notable because a purely analytic study should be perfectly replicable.³⁰⁹ When nine other scientists (including two well-reputed biomechanical engineers whom Bandak himself cited in his original paper) attempted to repeat Bandak's mathematics, they found numerous gross errors.³¹⁰ When these scientists repeated the computations themselves, they found that the correct value for every single neck force was at least ten times lower than the values reported.³¹¹ They determined that the corrected values *do not* exceed the threshold for neck injury.³¹² Confirming Bandak's errors, a second group of scientists independently attempted to replicate Bandak's work and produced results identical to the first.³¹³

In his response, Bandak admitted that the reported values did not result from the equations published in the paper.³¹⁴ Bandak suggested instead that he “basically integrated the [AHT] accelerations over the time duration of shaking,”³¹⁵ without providing any of the equations, data, or assumptions necessary to replicate the work.³¹⁶ So, when asked to produce a single “worked example” demonstrating how the reported forces could be computed, Bandak failed to do so.

Replication is a fundamental mechanism by which scientific validity is achieved. A work that cannot be replicated isn't bad science—it isn't science at all. Bandak's suggestion that

³⁰⁶ Duncan, *supra* note 254, at 763.

³⁰⁷ Rangarajan & Shams, *supra* note 257, at 281.

³⁰⁸ Laura K. Brennan et al., *Neck Injuries in Young Pediatric Homicide Victims*, 3 J. NEUROSURGERY PEDIATRICS 232, 235 (2009) (finding that 71% of children dying of AHT had injuries to the cervical spine or cord).

³⁰⁹ In other words, doing the same mathematics on the same, previously published, numbers should yield the same results regardless of who does it. In science, it is an author's burden to describe the experiments in sufficient detail that they can be repeated by an experienced investigator using only the text of the published paper.

³¹⁰ Susan Margulies et al., *Letter to the Editor: Shaken Baby Syndrome: A Flawed Biomechanical Analysis*, 164 FORENSIC SCI. INT'L 278, 278 (2006).

³¹¹ *Id.* at 278.

³¹² *Id.* at 279.

³¹³ Rangarajan & Shams, *supra* note 257, at 280.

³¹⁴ Faris A. Bandak, *Response to the Letter to the Editor*, 164 FORENSIC SCI. INT'L, 282, 282 (2006).

³¹⁵ *Id.* at 282.

³¹⁶ This author does not understand what this statement means in a rigorous, mathematical sense. However, since Bandak does not specify even the length or frequency of shaking used in his computations, no scientist will be able to replicate his result.

shaking sufficient to cause injury would necessarily cause neck injury is a prime example of how invalid biomechanical data have been misused in court.³¹⁷ Further reliance upon these data or citation to this work should be avoided.

Question 3: Has biomechanics shown that skull fractures are likely to result from a short fall?

As discussed in the Accident section above, short-distance falls are a common presenting history in children thought to be physically abused.³¹⁸ Several studies have demonstrated that the fetal and infant cranial bone increases in stiffness with age.³¹⁹ Coats and Margulies studied the material properties of infant cranial bone and suture in order to better predict the outcome of infant falls.³²⁰ They obtained donated skull and suture materials from 23 fetuses and infants ranging from 21 weeks of gestation to 13 months of age.³²¹ The authors measured the elastic modulus³²² in infants and found that the adult cranial bone modulus was 30 times higher (less deformable) than a 1 month old infant's.³²³ A 1 year old's cranial bone modulus was 18 times higher (less deformable) than a 1 month old infant's.³²⁴ Additionally, the pediatric suture³²⁵ deforms 30 times more than pediatric skull bone and 243 times more than adult bone before rupture.³²⁶ These large strains in the pediatric skull and suture may result in large-scale deformation upon impact and explain the diminished frequency of intracranial injury with short falls.

³¹⁷See *Cavazos v. Smith*, 565 U.S. 1, 5 (2011) (per curiam) (Ginsburg, J., dissenting).

³¹⁸A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger Than 2 Years of Age*, 90 PEDIATRICS 179, 182 (1992).

³¹⁹Timothy J. Kriewall, *Structural, Mechanical, and Material Properties of Fetal Cranial Bone*, 142 AM. J. OBSTETRICS GYNECOLOGY 707, 707 (1982); Gregg K. McPherson & Timothy J. Kriewall, *The Elastic Modulus of Fetal Cranial Bone: a First Step Towards Understanding of the Biomechanics of Fetal Head Molding*, 13 J. BIOMECHANICS 9, 14 (1980); Brittany Coats & Susan S. Margulies, *Material Properties of Human Infant Skull and Suture at High Rates*, 23 J. NEUROTRAUMA 1222, 1222-32 (2006).

³²⁰See Coats & Margulies, *supra* note 219.

³²¹*Id.*

³²²See Kriewall, *supra* note 271 (The "elastic modulus" is a ratio of stress to strain, and is constant for any uniform material. The smaller the elastic modulus, the more readily deformable it is).

³²³See Coats & Margulies, *supra* note 219.

³²⁴*Id.*

³²⁵The "suture" is the area of incomplete fusion of the cranial bone. It permits growth of the cranial bone and the intracranial structures.

³²⁶See Coats & Margulies, *supra* note 219.

Weber, a German pathologist, conducted two studies on infant cadavers in order to see how frequently fractures would occur on a variety of surfaces at changing table height.³²⁷ In the first study, the cadavers of 15 infants, no older than 8.2 months of age, were dropped (5 each) from 82 centimeters (32 inches) onto a stone-tile floor, a carpeted floor, and linoleumbacked with foam flooring.³²⁸ All of the infants had pathologic conditions that did not involve the head, and no skull fractures existed before testing, neither on palpation nor on skull radiographs.³²⁹ All the subjects made simultaneous contact with their back and parieto(side)-occipital(back) region of the head.³³⁰ All 15 of the cadavers sustained fractures and, in 3 cases, linear fractures crossed suture lines.³³¹

In his second paper, Weber tested an additional 35 cadavers with age ranges from newborn to 9.1 months at time of death.³³² Ten were dropped in the same manner and at the same height (32 inches) onto a foam mat 2.0 centimeters thick and 25 were dropped onto a doubly folded blanket 8 centimeters thick.³³³ One of the 10 (10%) infants dropped onto the rubber mat sustained parietal (side of the head) fractures that did not cross sutures.³³⁴ However, 4 out of 25 (16%) infants dropped onto the blanket sustained parietal skull fractures.³³⁵

However, in contrast to Weber's data, Snyder et al. found different results.³³⁶ Using newspaper clippings to screen for cases of free-falls in a six-state area, investigators were sent out to verify that the falls were, in fact, free-falls unimpeded from reaching the landing site by intervening obstacles and that the landing site had not changed since the time of the fall (e.g., construction or destruction of a building, change in soil or sand consistency due to significant rains).³³⁷ One hundred and ten free-fall cases were thoroughly investigated (age, biometrics,

³²⁷ See W. Weber, *Experimentelle Untersuchungen zu Schädelbruchverletzungen des Säuglings* [Experimental Study of Skull Fractures in Infants], 92 ZEITSCHRIFT FÜR RECHTSMEDIZIN 87 (1984); W. Weber, *Zur Biomechanischem Fragilität des Säuglingsschädels* [Biomechanical Fragility of Skull Fractures in Infants], 94 ZEITSCHRIFT FÜR RECHTSMEDIZIN 93 (1985).

³²⁸ W. Weber, *Experimental Study of Skull Fractures in Infants*, *supra* note 1 at 90. Five cadavers were dropped on each type of flooring surface. *Id.*

³²⁹ *Id.* at 89-91.

³³⁰ *Id.* at 91.

³³¹ *Id.* at 90-91.

³³² W. Weber, *Biomechanical Fragility of Skull Fractures in Infants*, *supra* note 1 at 100.

³³³ *Id.* at 94.

³³⁴ *Id.* at 94-95.

³³⁵ *Id.* at 95.

³³⁶ See RICHARD G. SNYDER ET AL., *Study of Impact Tolerance Through Free-Fall Investigations: Final Report*, HIGHWAY SAFETY RESEARCH INST., UNIV. OF MICH. (1977).

³³⁷ *Id.* at 11-14, 38.

height of fall, landing surface, initial landing posture, and medical outcome).³³⁸ Twelve cases were subsequently simulated in detail, using a computer simulation (MVMA 2-D Crash Victim Simulator, Version 3) that had been validated.³³⁹ Seven of the 12 simulations involved head-first impacts; among these were 5 children who were younger than 4 and who fell 10'6"-34'2".³⁴⁰ All of these children had skull fractures, concussion, or both.³⁴¹ Upon examination of injuries as a function of impact surface for head-first fall cases, only one child who fell from a height of approximately 12' did not have a skull fracture.³⁴² Snyder concluded that the tolerances for skull fracture in infants and toddlers without pathology were between 4-10 feet (i.e. after a 4-foot fall some would have a fracture, and after a 10-foot fall virtually all would have a skull fracture).³⁴³

Bertocci et al. used an anthropometric model to simulate a 3-year-old child rolling off a 27-inch "bed" onto playground foam, carpet, linoleum, and wood.³⁴⁴ Despite acknowledging "a paucity of injury criteria for children," Bertocci concluded that the risk of a contact head injury (essentially a skull fracture) resulting from a short rolling fall is "low."³⁴⁵

Coats and Margulies conducted a finite element model (FEM) study using parametric simulations of occipital impacts to predict the likelihood of occipital or parietal skull fractures.³⁴⁶ The authors found that elements arrays one standard deviation above the mean ultimate stress of occipital or parietal bone gave an 84.1% chance of occipital or parietal fracture.³⁴⁷ At three standard deviations above the mean ultimate stress of the parietal bone, there was a 99.8%

³³⁸ *Id.* at 38, Appx. A.

³³⁹ *Id.* at 20, 23-32, 76, 126.

³⁴⁰ *Id.* at 78.

³⁴¹ *Id.*

³⁴² *Id.* at 81.

³⁴³ *Id.* at 120-21. In another study, Snyder et al. abstracted 954 cases of children aged one to twelve years in which fall height, severity of injury, and fall surface (steel or concrete) were known from a database maintained by the FAA of 31,530 adult and child free-falls (fatal and non-fatal). See RICHARD G. SNYDER, *Impact Tolerances of Infants and Children in Free*, HIGHWAY SAFETY RESEARCH INST., UNIV. OF MICH., AAAM CONFERENCE (1970). Of those 954 cases, the authors took thirty-four cases in which biometric testing of the patient and full characterization of the fall and landing zone could be performed. *Id.* The LD₅₀ (the height at which roughly 50% die) occurred between forty-one and fifty feet. *Id.* According to Snyder's data, with lower free-falls came lower fatality rates and the lower rates of serious or critical injuries. *Id.* There were no deaths in falls of zero to five foot free falls onto steel or concrete in this series. *Id.*

³⁴⁴ Gina E. Bertocci et al., *Using Test Dummy Experiments to Investigate Pediatric Injury Risk in Simulated Short-Distance Falls*, 157 ARCHIVES PEDIATRICS ADOLESCENT MED. 480, 481-82 (2003).

³⁴⁵ *Id.* at 482-83.

³⁴⁶ Brittany Coats et al., *Parametric Study of Head Impact in the Infant*, 51 STAPP CAR CRASH J. 1, 1-2 (2007).

³⁴⁷ *Id.* at 5.

likelihood that the parietal bone would fracture.³⁴⁸ They found such a condition at the equivalent of an “82 cm fall onto concrete” and concluded that they had good validation with Weber (1984, 1985).³⁴⁹

The accident literature quoted earlier also suggests that short falls can result in skull fractures but at rates far less than those reported by Weber.³⁵⁰

Thus, the biomechanical literature demonstrates that short falls can result in skull fractures. However, in spite of Weber’s data, it would be unreasonable to suggest that all, or even a majority, of short falls cause skull fractures because multiple, well-designed clinical studies document that the vast majority of short falls do not result in a skull fracture.

Question 4: Has biomechanics shown that SDHs are likely to result from a short fall?

The Prange study mentioned above simulated a child being dropped onto concrete, carpet, and a foam mattress.³⁵¹ For the drop experiments, the model was suspended at 0.3 meters (1 foot), 0.9 meters (3 feet), and 1.5 meters (5 feet) over the landing surface.³⁵² The model was suspended with the head slightly below the remainder of the body, such that the head would strike the ground first.³⁵³ The investigators concluded that 0.3-meter falls were unlikely to cause subdural hematomas but were unable to comment on the likelihood of injury in the 0.9-meter and 1.5-meter falls due to uncertainty about injury tolerances.³⁵⁴

Thompson et al. simulated a 12-month-old child falling feet first onto wood, carpet, playground foam, linoleum over wood, and linoleum over concrete.³⁵⁵ They simulated falls from 0, 9, and 29 inches, measured from the dummy’s feet.³⁵⁶ The authors concluded “the risk of severe head injury for a 12-month-old child in feet-first free falls across all tested surfaces and heights was low.”³⁵⁷ A similar study by some of the same authors comparing falls onto wet

³⁴⁸ *Id.* at 8.

³⁴⁹ *Id.* at 2, 8, 11.

³⁵⁰ See Lyons & Oates, *supra* note 54, at 126-27; Helfer, *supra* note 51, at 534-35; Nimityongskul, *supra* note 58, at 185-86.

³⁵¹ Prange, *supra* note 235, at 144-45.

³⁵² *Id.* at 145.

³⁵³ *Id.*

³⁵⁴ *Id.*

³⁵⁵ Angela K. Thompson et al., *Assessment of Head Injury Risk Associated with Feet-First Free Falls in 12 Month-Old Children Using an Anthropomorphic Test Device*, 66 J. TRAUMA INFECTION & CRITICAL CARE 1019, 1020 (2009).

³⁵⁶ *Id.*

³⁵⁷ *Id.* at 1028.

linoleum to dry linoleum found similarly low risks for femur (large bone in the upper part of the leg) fracture or head injury.³⁵⁸

In another study, Thompson et al. visited the homes of children who presented to the hospital for treatment after an accidental short fall.³⁵⁹ The investigators specifically excluded cases where there was concern for possible child abuse.³⁶⁰ Similar to prior studies, the experimenters found serious injuries to be rare and found no critical or life-threatening injuries.³⁶¹ Additionally, the surface the child fell on, pre-fall position, post-fall position, and motion prior to the fall all failed to demonstrate a statistically significant effect on injury severity resulting from the fall.³⁶²

The short fall reconstruction articles cited here represent a field in its infancy. They represent reconstructions of only a handful of seemingly limitless permutations of fall height, impact surface, initial position, fall biomechanics, and dummy characteristics that are involved in simulating a short fall. Ongoing and future research on the topic will most likely refine our current knowledge on the specific biomechanical parameters that will cause SDHs.

Question 5: Does the biomechanics literature show that shaking can cause retinal hemorrhages?

Rangarajan et al. constructed an FEM of an infant eye based on head CTs of six normal, young children.³⁶³ The model showed that rhythmic shaking significantly increases the stress on the retina.³⁶⁴ The strongest forces were found at the posterior pole and the periphery.³⁶⁵

³⁵⁸Ernest Deemer et al., *Influence of Wet Surfaces and Fall Height on Pediatric Injury Risk in Feet-First Freefalls as Predicted Using a Test Dummy*, 27 MED. ENGINEERING & PHYSICS 31, 38 (2005).

³⁵⁹See Thompson, *supra* note 86, at 144. Parents or guardians of children selected for case study had the option to allow the investigators to: 1) review the child's medical records, 2) interview the caregiver and review the child's medical records, or 3) investigate the fall scene at the home, interview the caregiver, and review the child's medical records. *Id.*

³⁶⁰*Id.*

³⁶¹*Id.* at 148.

³⁶²*Id.*

³⁶³Nagarajan Rangarajan et al., *Finite Element Model of Ocular Injury in Abusive Head Trauma*, 13 J. AM. ASS'N FOR PEDIATRIC OPHTHALMOLOGY & STRABISMUS 364, 365 (2009).

³⁶⁴*Id.* at 368.

³⁶⁵*Id.*

Notably, as Narang discussed in his first article, this is where retinal hemorrhages in AHT are often found.³⁶⁶

A similar model by Hans et al. demonstrated that force on the retina during a single shaking incident is about 13 times that of a short fall with a head impact.³⁶⁷ A 4-cycle shaking event produces forces on the retina 50 times that of a short fall.³⁶⁸ The forces on the retina exceeded the adhesive strength of adult monkey retinas for the shaking, but not the short fall simulations.³⁶⁹ These results coincide with clinical studies suggesting that retinal hemorrhages are more common in inflicted than accidental trauma.³⁷⁰

Despite these findings, some learned biomechanical engineers suggest caution in interpreting these FEM studies.³⁷¹ They note that material properties, tissue-tissue interactions, and injury tolerances have not been measured or published for the pediatric eye.³⁷² They comment that, as with all biomechanical studies, a finite element analysis is only as good as its inputs and “inaccuracy of these inputs will yield fallacious outputs.”³⁷³

Animal studies have wrought further data on this topic. Coats et al. subjected 3–5-day-old piglets to a single, abrupt head rotation using a mechanical apparatus.³⁷⁴ The piglets were kept alive for six hours, euthanized, and then autopsied.³⁷⁵ Out of 51 animals, 4 (8%) had retinal hemorrhages.³⁷⁶ Similarly, as mentioned above, Finnie et al. noted minor retinal hemorrhages in two of seven sheep who were manually shaken.³⁷⁷

In summary, as with biomechanical studies on short falls, the data are preliminary and limited but enlightening. The current state of knowledge is that biomechanical studies support clinical evidence that retinal hemorrhages can be caused by shaking.

³⁶⁶See Sandeep Narang, *A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome*, 11 Hous. J. Health L. & Pol’y 505, 548–58 (2011).

³⁶⁷Steven Alex Hans et al., *A Finite Element Infant Eye Model to Investigate Retinal Forces in Shaken Baby Syndrome*, 247 GRAEFES ARCHIVE FOR CLINICAL & EXPERIMENTAL OPHTHALMOLOGY 561, 567–68 (2009).

³⁶⁸*Id.* at 568.

³⁶⁹*Id.* at 568–70.

³⁷⁰See Narang, *supra* note 10, at 550–58.

³⁷¹See Susan Margulies et al., *What Can We Learn from Computational Model Studies of the Eye?*, 13 J. Am. Ass’n FOR PEDIATRIC OPHTHALMOLOGY & STRABISMUS 332, 332 (2009).

³⁷²*Id.*

³⁷³*Id.*

³⁷⁴Brittany Coats et al., *Ocular Hemorrhages in Neonatal Porcine Eyes from Single, Rapid Rotational Events*, 51 INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCI. 4792, 4792 (2010).

³⁷⁵*Id.* at 4793.

³⁷⁶*Id.* at 4794.

³⁷⁷Finnie, *supra* note 242, at 237, 239.

Conclusion

Scientists have a peculiar fondness for data. Data are the recorded results of the most fundamental scientific skill: careful observation. Good science welcomes *valid* data of all kinds and from all disciplines—dropping test dummies in a lab, shaking immature animals, and counting injured children as they come through the ER. The wise scientist recognizes that all data are flawed in some way. However, data are not discarded upon the identification of a single flaw. It is assessed in totality while carefully balancing the limitations of those data and, consequently, according appropriate weight to the compilation of all data. The biomechanical literature discussed here is in its infancy and, in spite of its flaws, is useful and informative. For anyone to assert blanket superiority of one type of data over another type (such as clinical data) is not just scientifically irresponsible; it is scientifically arrogant.

Hypoxia

As noted earlier, one of the alternative explanations proposed for the findings seen in victims of AHT is “hypoxia.”³⁷⁸ Broadly, the term “hypoxia” is used to refer to a low level of oxygen in the body.³⁷⁹ As a plastic condition, hypoxia’s impact on the body depends upon a number of factors,³⁸⁰ but broadly, it is important to think of hypoxia as both magnitude and duration. A small amount of hypoxia (slightly lower level of oxygen in the body) for a long duration has a very different effect on the body than profound hypoxia for a short duration.³⁸¹ It is important to recognize that “hypoxia” is not a single clinical or physiological entity and to speak of it as such is at best imprecise, and at worst simply wrong. This must be borne in mind as we unpack the role of “hypoxia” as a potential explanation for the finding seen in AHT.

For the past decade, some authors have proposed that “thin film” SDH, retinal hemorrhages, and acute encephalopathy (brain injury) can all be explained as being caused by

³⁷⁸ needs citation

³⁷⁹ Oxygen is carried in the blood from the lungs to the body and organs by two main mechanisms. The vast majority of oxygen in the blood is bound to hemoglobin (a protein in red blood cells) with a smaller percentage of oxygen actually dissolved in the blood.

³⁸⁰ needs citation

³⁸¹ needs citation

hypoxia alone, absent trauma.³⁸² Evolving from the hypoxia theory have been two adjunct hypotheses: (1) “dysphagic” choking³⁸³ and (2) cervical spine (neck) injury³⁸⁴ as causative of hypoxia, and thus, of the findings seen in AHT. We will now review the published reports and clinical data used to support these proposals, often referred to as the “Unified Theory.”³⁸⁵ It is important to note that in much of the literature used to support the hypoxia hypothesis, hypoxia as a clinical entity is undefined, making true analysis and comparison imprecise at best and misleading at worst.

A. Geddes and the Unified Theory

One of the cornerstones of the Hypoxia Hypothesis is a series of three papers by the British neuropathologist Dr. Jennian Geddes.³⁸⁶ These have been often characterized as “*Geddes 1*,”³⁸⁷ “*Geddes 2*,”³⁸⁸ and “*Geddes 3*.”³⁸⁹ Broadly, they are used to characterize the “Unified Theory” of hypoxia as the main (or sole) cause of the spectrum of findings associated with AHT. We will now describe them in some detail, as they are misunderstood by many and, thus, are often improperly cited.

Geddes 1: In 2000, Geddes and colleagues published a descriptive report on a non-sequential series of infant and child fatalities.³⁹⁰ Given the paucity of prior published literature on the topic, the authors sought to analyze and report upon the neuropathologic findings in 53

³⁸²J.F. Geddes et al., *Dural Haemorrhage in Non-Traumatic Infant Deaths: Does it Explain the Bleeding in ‘Shaken Baby Syndrome’?* 29 NEUROPATHOLOGY & APPLIED NEUROBIOLOGY 14, 18-19 (2003).

³⁸³See D. G. Talbert, *Paroxysmal Cough Injury, Vascular Rupture and ‘Shaken Baby Syndrome,’* 64 MEDICAL HYPOTHESES 8, 8-13 (2005); D. G. Talbert, *Dysphagia as a Risk Factor for Sudden Unexplained Death in Infancy,* 67 MEDICAL HYPOTHESES 786 (2006); J.F. Geddes & D.G. Talbert, *Paroxysmal Coughing, Subdural and Retinal Bleeding: A Computer Modelling Approach,* 32 NEUROPATHOLOGY & APPLIED NEUROBIOLOGY 625 (2006); Patrick D. Barnes et al., *Infant Acute Life-Threatening Event--Dysphagic Choking Versus Nonaccidental Injury,* 17 SEMINARS IN PEDIATRIC NEUROLOGY 7 (2010).

³⁸⁴Evan W. Matshes et al., *Shaken Infants Die of Neck Trauma, Not of Brain Trauma,* 1 ACAD. FORENSIC PATHOLOGY 82, 83-86 (2011).

³⁸⁵needs citation

³⁸⁶See J.F. Geddes et al., *Neuropathology of Inflicted Head Injury in Children I. Patterns of Brain Damage,* 124 BRAIN 1290 (2001); J.F. Geddes et al., *Neuropathology of Inflicted Head Injury in Children II. Microscopic Brain Injury in Infants,* 124 BRAIN 1299 (2001); J.F. Geddes et al., *supra* note X, at 14.

³⁸⁷J.F. Geddes et al., *supra* note X, at 1290.

³⁸⁸J.F. Geddes et al., *supra* note X, at 1299.

³⁸⁹See Geddes et al., *supra* note 305.

³⁹⁰See Geddes et al., *supra* note 308

cases of infants and children who had suffered inflicted brain injury.³⁹¹ They identified this cohort of fatalities using very similar criteria,³⁹² which many other authors and investigators have in the past and still currently utilize.³⁹³ The investigators were not blinded to the cause or manner of death, nor were they blinded to the neuropathologic findings.³⁹⁴ All clinical and legal records were reviewed and all brains were similarly systematically sampled and stained.³⁹⁵

Of these 53 subjects, 37 were infants (under 1 year of age).³⁹⁶ Half (n=27, 51%) had significant extracranial injuries e.g., burns, bruising, and/or fractures.³⁹⁷ The majority (n=45, 85%) had signs of impact, including 19 (36%) with skull fractures.³⁹⁸ The authors reported that 44 (81%) had subdural hemorrhage (SDH) with 34 being “thin film” (which was not defined by the authors).³⁹⁹ Of the 38 subjects in which a pathologist examined the eyes, the authors report that 27 (71%) had retinal hemorrhages.⁴⁰⁰ When compared with those without SDH, the authors reported that the presence of RH was statistically significantly associated with the presence of SDH ($p < 0.001$).⁴⁰¹ The authors reported that of the ten subjects without SDH, five did not have RH.⁴⁰² However, they also reported that they examined the retinas in only half of the subjects without SDH.⁴⁰³

In eight subjects that the authors called “shaken-only” by virtue of the absence of findings of cranial impact, five (of the six with examined eyes) had RH and seven presented with

³⁹¹*Id.* at 1290.

³⁹²The authors identified AHT cases either: 1) by confession; 2) by criminal conviction, with or without the presence of extracranial injuries on the child; 3) by cases without conviction in which unexplained injuries to the rest of the child’s body, in addition to head injury, were present; or, 4) by cases in which there was a “major discrepancy” between the injury explanation given by the caregiver and “significant injuries” or “the history was developmentally incompatible” with the injury. *Id.* at 1290-91.

³⁹³ needs citation

³⁹⁴*Id.* at 1291-94.

³⁹⁵*Id.* at 1291. Although, one case had only a clinical history available for review, the investigators found “sufficient detail to merit [the case’s] inclusion in the study.” *Id.*

³⁹⁶*Id.*

³⁹⁷*Id.* at 1291-92.

³⁹⁸*Id.*

³⁹⁹*Id.* at 1292. The authors note that the “thin film” designation was used in post-mortem reports in which the SDHs found were “trivial in terms of quantity of blood.” *Id.*

⁴⁰⁰*Id.* at 1294.

⁴⁰¹*Id.*

⁴⁰²*Id.*

⁴⁰³*Id.* It is important to note that if the author examined the other five subjects without SDH and either four or five of them had RH, then the relationship would no longer be significant by chi square testing ($p=0.068$ or $p=0.209$). By not examining all of the eyes of their “control” group, they falsely report significance when the relationship may not be true.

collapse or respiratory arrest.⁴⁰⁴ The authors reported the most common microscopic finding was “global neuronal hypoxia-ischaemia,” seen in 84% of the infants and 63% of the older children.⁴⁰⁵ Only three (6%) of the subjects had diffuse axonal injury.⁴⁰⁶ The authors found no differences between the pathologies of subjects with and without evidence of impact.⁴⁰⁷ The authors reported three significant clinical differences between infants and older children in their cohort: 1) infants had more apnea,⁴⁰⁸ 2) infants had fewer extracranial (outside the skull)injuries,⁴⁰⁹ and 3) infants had less subscalpular bruising (evidence of head impact).⁴¹⁰

Geddes 2: In their second paper, the same authors selected the same 37 infants from their previous cohort, to which they added 14 “control” infants for comparison.⁴¹¹ The control group was a non-sequential group of infants who apparently died from non-abusive causes.⁴¹² The authors reported on the intracranial histology⁴¹³ of these subjects and compared them with the infants utilized in their earlier cohort.⁴¹⁴ Again, the authors were neither blinded to the clinical information nor the ultimate histopathologic findings.⁴¹⁵

The authors reported that, of the 37 cases of abusive head trauma (AHT), 25 (68%) had evidence of β APP staining (a stain conventionally associated with traumatic injury)⁴¹⁶ in axons.⁴¹⁷ None of the control infants were reported to have β APP staining identified in their brains.⁴¹⁸ They also reported that 29 (78%) of the cases of AHT had widespread hypoxic neuronal injury, while only 1 infant (7%) in the control group had histologic evidence of severe hypoxia.⁴¹⁹ As noted earlier, 21 infant cases of AHT (70% of those examined) had bilateral RH

⁴⁰⁴*Id.* at 1295-96.

⁴⁰⁵*Id.* at 1294.

⁴⁰⁶*Id.* at 1292.

⁴⁰⁷*Id.* at 1295-96.

⁴⁰⁸*Id.* at 1294.

⁴⁰⁹*Id.*

⁴¹⁰*Id.* at 1291-92.

⁴¹¹Geddes et al., *supra* note 312, at 1299-1300.

⁴¹²*Id.*

⁴¹³“Histology” means the microscopic evaluation of tissue at the cellular level.

⁴¹⁴Geddes et al., *supra* note 312, at 1300-1301.

⁴¹⁵*Id.* at 1299-1302.

⁴¹⁶See Geddes et al., *supra* note 305, at 1294; Michael W. Johnson et al., *Axonal Injury in Young Pediatric Head Trauma: A Comparison Study of β -Amyloid Precursor Protein (β APP) Immunohistochemical Staining in Traumatic and Nontraumatic Deaths*, 56 J. FORENSIC SCIS. 1198, 1198 (2011).

⁴¹⁷Geddes et al., *supra* note 312, at 1300.

⁴¹⁸*Id.*

⁴¹⁹*Id.* at 1300-01.

and 9 cases did not have RH.⁴²⁰ Seven cases were not examined.⁴²¹ The comparison group did not have their retinal findings described.⁴²²

The authors concluded that their “findings strongly suggest that severe traumatic axonal damage is a rarity in infant NAI unless there is considerable impact and that the diffuse brain damage responsible for loss of consciousness in the majority of cases is hypoxic rather than traumatic.”⁴²³

Geddes 3: Dr. Geddes and a similar set of co-authors then focused their attention to the dural covering of the brain.⁴²⁴ They assembled 50 non-sequential fetal, neonatal, and infants who had non-traumatic deaths.⁴²⁵ How this cohort was selected is not described.⁴²⁶ To this cohort of cases, the authors compared three selected cases of AHT.⁴²⁷ The authors did not describe how they chose these three infants, only that they were part of their earlier cohort.⁴²⁸ Once again, the authors were neither blinded to the clinical information nor to the histologic findings.⁴²⁹

Of the 50 subjects, 41 (82%) were either fetal or neonatal deaths, with 17 (34%) intrauterine deaths and 16 (33%) perinatal (younger than 7 days of life) deaths.⁴³⁰ Of the 50 cases, 26 (52%) died from hypoxia, 8 (16%) from infection (not defined) with severe hypoxia (not defined), and 6 (12%) from infection (not defined) without hypoxia.⁴³¹

Evaluation of the dural covering revealed only one subject (2%) with a macroscopic (seen without a microscope) SDH.⁴³² This infant was a 25-week gestation whose mother had chorioamnionitis (infected placenta) and died from sepsis (blood infection).⁴³³ The authors

⁴²⁰*Id.* at 1302.

⁴²¹*Id.*

⁴²²*See id.*

⁴²³*Id.* at 1304.

⁴²⁴*See* Geddes et al., *supra* note 308, at 15.

⁴²⁵*Id.*

⁴²⁶*See id.*

⁴²⁷*Id.* The authors labeled the comparison cases as “cases of classical ‘shaken baby syndrome.’” *Id.*

⁴²⁸*See id.*

⁴²⁹*Id.* at 15, 17-18.

⁴³⁰*Id.* at 15.

⁴³¹*Id.*

⁴³²*Id.*

⁴³³*Id.*

reported that 36 (72%) of the remaining cases had intradural hemorrhage (IDH).⁴³⁴ This is described as “bleeding inside the strips of dura.”⁴³⁵ Retinas were not examined.⁴³⁶

The authors *hypothesized* that the IDH noted in their sample was in essence, a precursor of the larger SDH,⁴³⁷ which would be typically associated with trauma.⁴³⁸ Despite reporting that there was no statistically significant relationship between hypoxia and IDH,⁴³⁹ the authors extrapolated that the microscopic IDH could be caused by hypoxia (alone or with other factors) and, thus, larger SDHs could be caused by hypoxia (with or without other factors) as well.⁴⁴⁰ Furthermore, despite no physiological data, the authors theorized that “cerebral venous hypertension and congestion, arterial hypertension and brain swelling, coupled with immaturity and hypoxia related vascular fragility” contributed to a cascade of events leading to findings similar to those seen in AHT.⁴⁴¹ Additionally, without reporting ophthalmologic findings, they hypothesized that RHs occur from a similar mechanism.⁴⁴²

B. Scientific critique of the Unified Theory

The series of papers by Geddes et al., attempted to address two main themes. *Geddes 1* and *Geddes 2* attempted to describe the histopathologic findings in infants and children who are victims of AHT. They asserted that the histopathologic findings associated with AHT are similar to that seen in hypoxia-associated deaths. *Geddes 3* attempted to describe how SDH can occur in the absence of trauma, utilizing hypoxia as the primary culprit. Clearly, the use of fetal and perinatal deaths obscures any conclusions on IDH or SDH because these are common findings at baseline (as outlined below).

Given the unblinded nature of the *Geddes 1* and *Geddes 2* subjects, true interpretation of the implications of the results must be guarded. A recent study of higher and more rigorous methodology involved three parallel assessors who were blinded to the clinical information.⁴⁴³

⁴³⁴*Id.*

⁴³⁵*Id.*

⁴³⁶*See id.* at 15, 17-18.

⁴³⁷*Id.* at 19.

⁴³⁸*Id.* at 14.

⁴³⁹*See id.* at 17.

⁴⁴⁰*Id.* at 19-20.

⁴⁴¹*Id.* at 19.

⁴⁴²*Id.* at 19-20.

⁴⁴³Johnson et al., *supra* note 40, at 1199-1200.

The subjects were 24 child fatalities from a variety of causes (including AHT).⁴⁴⁴ The reviewers were asked to assess the histopathology (β -APP) and indicate any evidence of trauma.⁴⁴⁵ These blinded and independent assessors rated 5 of the 7 child homicides as “trauma” and 15 of the 17 controls as “non-trauma.”⁴⁴⁶ This indicates that while not perfect, the histopathologic findings are exceedingly informative in determining the presence or absence of trauma. The authors rightly indicated that β -APP is clearly associated with trauma but urged caution saying, “the utility of β -APP is quite powerful if not confounded by global hypoxic-ischemic injury, [and] ultimately, β -APP studies should be only one piece of information in the determination of cause and manner of death.”⁴⁴⁷

Another notable aspect of the *Geddes 1* and *2* papers is how their subjects were identified. The authors used inclusion criteria which included confession, conviction (with or without additional injuries), and discrepancy between findings and history provided (without adjudication).⁴⁴⁸ Criteria like these have been utilized for decades by many other researchers.⁴⁴⁹ It is scientifically and logically inconsistent for some authors to criticize these other papers as having flawed inclusion methodology and yet cite the *Geddes* papers as “having important clinical implications”⁴⁵⁰ or “landmark.”⁴⁵¹ If the conclusions of the *Geddes* papers are sound and meaningful, then so are the conclusions of other studies which utilize the same methodology. One cannot have it both ways.

With regards to *Geddes 3*, there are two major obstacles in interpreting this paper. First, the authors present clinical information and data with significant imprecision. For example,

⁴⁴⁴*Id.* at 1199.

⁴⁴⁵*Id.*

⁴⁴⁶*Id.* at 1201.

⁴⁴⁷*Id.* at 1198.

⁴⁴⁸ *Geddes et al.*, *supra* note 305, at 1290-91; *Geddes et al.*, *supra* note 308, at 1299.

⁴⁴⁹ See A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age*, 2 PEDIATRICS 179 (1992); Linda Ewing-Cobbs et al., *Neuroimaging, Physical, and Developmental Findings After Inflicted and Noninflicted Traumatic Brain Injury in Young Children*, 102 PEDIATRICS 300 (1998); K.W. Feldman et al., *The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study*, 108 PEDIATRICS 636 (2001); K.P. Hymel et al., *Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma During Infancy: Results of a Prospective, Multicentered, Comparative Study*, 119 PEDIATRICS 922 (2007); S. Maguire et al., *Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review*, 94 ARCHIVES OF DISEASE IN CHILDHOOD 860, 860-867 (2009).

⁴⁵⁰ Squier W. *Shaken Baby Syndrome: The Quest for Evidence*, 50 DEV. MED. CHILD 10, 10-14 (2008).

⁴⁵¹ Barnes PD. *Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine*, 49 RADIOLOGIC CLINICS N. AM, 205, 205-229.

indicating that an infant died from an “infection,” or simply had “hypoxia,” without clinical specificity leaves these terms vague and uninterpretable. “Infection” could be meningitis,⁴⁵² sepsis,⁴⁵³ pneumonia, or pyelonephritis⁴⁵⁴; all of which are distinctly different medical entities. “Hypoxia,” as noted above, is a heterogenous clinical designation that could be profound or trivial. Without clinical parameters, one cannot interpret the implications or potential signs or symptoms of the hypoxia.

Second, none of the authors’ findings are new. The presence of intradural hemorrhage⁴⁵⁵ (IDH) associated with fetal demise has been known for decades, if not centuries.⁴⁵⁶ The authors themselves, citing Chase,⁴⁵⁷ note “early study documented intradural bleeding as a ‘constant finding’ in premature infants.”⁴⁵⁸ In fact, one of the seminal monographs on birth-related injuries by Schwartz⁴⁵⁹ from 1961 reports this history in the study of intracranial findings in the neonate. In summarizing the understanding of findings seen in fetuses and neonates, Schwartz writes “hemorrhage affects the dural reduplications (falx and tentorium) rather frequently.”⁴⁶⁰ In this context, the Geddes et al. report of IDH in a cohort of mostly fetal and perinatal deaths is neither new nor illustrative.

The presence of macroscopic, radiographically apparent SDH due to birth is also a well-described finding. An atlas by Cruveilhier in 1831⁴⁶¹ contains drawings of extensive meningeal hemorrhage over the hemispheres and, according to Schwartz,⁴⁶² Cruveilhier reported that these hemorrhages occurred in “at least one-third of neonatal deaths”. This is similar to the frequency (~ 25%) with which SDH has been reported in the past decade on neuroimaging of healthy term

⁴⁵² “Meningitis” indicates an infection or inflammation of the meninges (the outer covering of the brain).

⁴⁵³ “Sepsis” is a potentially deadly condition characterized by whole body inflammation and organ dysfunction, usually caused by an infection.

⁴⁵⁴ “Pyelonephritis” means infection or inflammation of the kidney.

⁴⁵⁵ “Intradural” hemorrhage means inside the dura, or not macroscopically apparent on the surface of the brain.

⁴⁵⁶ See William H. Chase, *An Anatomical Study of Subdural Haemorrhage Associated with Tentorial Splitting in the Newborn*, 51 SURGERY GYNECOLOGY OBSTETRICS 31 (1930). PHILIP SCHWARTZ & EADELY L. HOLLAND, BIRTH INJURIES OF THE NEWBORN: MORPHOLOGY, PATHOGENESIS, CLINICAL PATHOLOGY AND PREVENTION (S. Karger ed., 1961). Schwartz reports that intracranial hemorrhage was first described in 1804. Schwartz outlines the continued study of falcine and tentorial hemorrhage (different sections of the dura) in newborns for over 100 years.

⁴⁵⁷ *Id.*

⁴⁵⁸ See Geddes et al, *supra* note 305.

⁴⁵⁹ See Schwartz, *supra* note 332.

⁴⁶⁰ *Id.* at 38.

⁴⁶¹ Jean Cruveilhier, Paper presented at Confer. a l’occasion de la distribution des prix aux élèves sages-femmes de la Maison d’Accouchement de Paris, (June 23, 1831) (Fr.).

⁴⁶² See Schwartz, *supra* note 332.

neonates.⁴⁶³ It is striking that Geddes et al. reported only 1 (2%) subject with gross SDH. This rate is statistically different from the one-third reported in the mid 1800s or the one-quarter reported in the 21st century (chi square, $p=0.000$ and $p=0.001$).⁴⁶⁴ This calls into question whether the entire cohort used by Geddes et al. in *Geddes 3* is systematically different and not representative at all of “typical cases,” either living or dead.

Lastly, and most importantly, if this model of hypoxia as outlined by Geddes et al. is indeed a reasonable explanation for an SDH similar to the ones associated with AHT, why were they not seen in their own study? The only SDH reported was in a fetus that did not have hypoxia, but instead sepsis, reported as a cause of death. In their study, all infants with hypoxia identified as a factor in their death did not have an SDH. If this model had fidelity to a true underlying pathophysiologic process, it would be expected that the finding they are attempting to explain would be present in at least some of the cases; it was present in none.

To truly assess whether hypoxia alone can result in SDH, one must use subjects which are more reflective of victims of AHT.⁴⁶⁵ Infant and child victims of drowning or near drowning represent a nearly ideal population. The insult is pure hypoxia, the ages are similar, the event is (sadly) common, the timing of the event is usually clear and not under dispute, and the outcome can be either fatal or non-fatal.

Rafaat and colleagues reviewed all infants and children admitted to the Children’s Hospital of San Diego over a 17-year period who were on a drowning registry.⁴⁶⁶ Of the 961 infants or children identified, 156 had a head CT scan within 24 hours of admission (some having 2 scans). Sixty-one were under three years of age, which was similar to other epidemiologic studies on drowning injuries. Fifty-eight scans had an abnormality identified. *None* of the abnormal scans

⁴⁶³ See V. J. Rooks et al., *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, 29 AMER. J. NEURORADIOLOGY 1082 (2008); E. H. Whitby et al., *Frequency and Natural History of Subdural Haemorrhages in Babies and Relation to Obstetric Factors*, 362 LANCET 846 (2003).

⁴⁶⁴ See Cruveilhier, *supra* note 337, and Schwartz, *supra*, note 332. Of note, when reporting a p-value to three decimal points, any value below 0.0005 is simply reported as 0.000. Thus, a 0.0006 would be reported as 0.001, but 0.0003 would be 0.000.

⁴⁶⁵ In a subsequent study, Dr. Geddes’ colleagues attempted to demonstrate hypoxia-associated SDH’s, but utilized a cohort of patients that were fetuses (as premature as 26 weeks) and young neonates (the oldest being only 19 days). See also Marta C. Cohen & Irene Scheimberg, *Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: an Observational Study from Two Referral Institutions in the United Kingdom*, 12 PEDIATRIC DEV. PATHOLOGY 169 (2009).

⁴⁶⁶ Karim T. Rafaat et al., *Cranial Computed Tomographic Findings in a Large Group of Children with Drowning: Diagnostic, Prognostic, and Forensic Implications*, 9 PEDIATRIC CRITICAL CARE MED. 567 (2008).

had any SDH noted.⁴⁶⁷ Thus, out of 156 children with a spectrum of hypoxia insults and clinical outcomes (including 41 deaths), *none* had an SDH on the head CT scan. This would mean that, at most, hypoxia causes SDH 2% of the time, but it may be as rare as never.⁴⁶⁸ This result accorded with Taylor et al.'s radiologic study in 1985⁴⁶⁹ and Byard's⁴⁷⁰ and Hurley's⁴⁷¹ pathology studies in 2007 and 2010, respectively.

Additionally, multiple lines of research have demonstrated that hypoxia is not a putative factor in causing RHs. Pitetti et al. prospectively studied 128 children less than 2 years of age who presented with apparent life threatening events (ALTEs) to determine the presence of RHs.⁴⁷² Seventy-three of the 128 (57%) children received dilated fundoscopic exams. Only 1 child (1.4%) had RHs.⁴⁷³ Upon further investigation, that case was determined to be a case of confessed abuse. Odom et al. prospectively examined the prevalence and character of RHs in patients in a pediatric ICU who had received at least one minute of chest compressions and survived.⁴⁷⁴ After using strict exclusion criteria (such as excluding patients that had evidence of trauma, documented retinal hemorrhages before CPR, or suspicion of child abuse), Odom et al. found 43 patients who met criteria for their study. Of the 43 patients, "[t]he mean duration of chest compressions was 16.4 minutes . . . with 58% lasting between 1 and 10 minutes."⁴⁷⁵ All patients survived, and the authors found small punctate retinal hemorrhages in only 1 patient

⁴⁶⁷ *Id.*

⁴⁶⁸ The arrival at the upper limit of 2% is analogous to a "margin of error" in statistics. In statistical analysis, the 2% is the largest outside chance that these findings would be present if the study were large enough to include all possible cases of drowning.

⁴⁶⁹ Steven B. Taylor et al., *Central Nervous System Anoxic-Ischemic Insult in Children Due to Near-Drowning*, 156 PEDIATRIC RADIOLOGY 641 (1985).

⁴⁷⁰ Roger W. Byard et al., *Lack of Evidence for a Causal Relationship Between Hypoxic-Ischemic Encephalopathy and Subdural Hemorrhage in Fetal Life, Infancy, and Early Childhood*, 10 PEDIATRIC DEV. PATHOLOGY 348, 348 (2007).

⁴⁷¹ M. Hurley, et al., *Is There a Causal Relationship Between the Hypoxia-Ischaemia Associated with Cardiorespiratory Arrest and Subdural Haematomas? An Observational Study*, 83 BRITISH J. RADIOLOGY 736, 736–37 (2010).

⁴⁷² Raymond D. Pitetti et al., *Prevalence of Retinal Hemorrhages and Child Abuse in Children Who Present with an Apparent Life-Threatening Event*, 110 PEDIATRICS 557 (2002).

⁴⁷³ *Id.*

⁴⁷⁴ Amy Odom et al., *Prevalence of Retinal Hemorrhages in Pediatric Patients After In-Hospital Cardiopulmonary Resuscitation: A Prospective Study*, 99 PEDIATRICS e3 (1997).

⁴⁷⁵ *Id.*

(2.3%). No patient had severe RHs. Finally, numerous animal studies investigating the effect of hypoxia on the retina have failed to demonstrate RHs.⁴⁷⁶

C. Adjunct hypotheses:

There are at least two parallel hypotheses to hypoxia that have recently been proposed. These hypotheses are “dysphagia/choking” or “coughing” and “neck injury.” The “dysphagia/choking”/“coughing” hypothesis proposes that when an infant or a child coughs or gags, there is an increase in cerebral (brain) vascular pressure. This increased pressure, in the presence of hypoxia, causes rupture of cerebral blood vessels.

Initially proposed by Talbert⁴⁷⁷ in a non-peer-reviewed journal, *Medical Hypotheses*, it has since been promulgated by a few other authors.⁴⁷⁸ Talbert has proposed that SDHs result from hypoxia and choking/coughing related to pertussis,⁴⁷⁹ pyloric stenosis (stomach obstruction),⁴⁸⁰ and/or gastroesophageal reflux with choking.⁴⁸¹ Talbert and Geddes subsequently attempted to replicate these physiologic conditions utilizing a mathematical software model of an infant.⁴⁸² Talbert and Geddes asserted that their model “has supported clinical observations, showing that the conditions necessary for subdural and retinal bleeding do occur in paroxysmal coughing, although it cannot prove that the bleeding is necessarily or even actually present clinically.”⁴⁸³

⁴⁷⁶ Charanjit Kaur et al., *Early Response of Neurons and Glial Cells to Hypoxia in the Retina*, 47 INVESTIGATIVE OPHTHAMOLOGY VISUAL SCI. 1126 (2006) (no RHs in rats with hypoxia of the retina); Taiji Nagaoka et al., *The Effect of Nitric Oxide on Retinal Blood Flow During Hypoxia in Cats*, 43 INVESTIGATIVE OPHTHAMOLOGY VISUAL SCI. 3037 (2002) (decreased retinal blood flow leads to vessel dilation but no RHs in cats).

⁴⁷⁷ See *supra* note 221. Talbert initially proposed that coordinated coughs can cause a cascade of increased systemic arterial pressure which was beyond the threshold of cerebral blood vessels. This increased blood pressure would then lead to SDH and RH. Talbert identified Pertussis (Whooping Cough) as a “natural experiment” of this phenomenon.

⁴⁷⁸ See Geddes, *supra* note 3, and Findley et al, *supra* note 3; see also Mudher Al-Adnani, Marta C. Cohen, Irene Scheimberg, *Gastroesophageal Reflux Disease and Sudden Infant Death: Mechanisms Behind an Under-Recognized Association*, 14 PEDIATRIC DEV. PATHOLOGY 53 (2010).

⁴⁷⁹ See Talbert, *supra* note 383.

⁴⁸⁰ D.G. Talbert, *Pyloric Stenosis as Cause of a Venous Hypertensive Syndrome Mimicing True Shaken Baby Syndrome*, 1 J. TRAUMA TREATMENT 1, 1, 5, 8 (2012).

⁴⁸¹ D.G. Talbert, *Cyclic Vomiting Syndrome: Contribution to Dysphagic Infant Death*, 73 MED. HYPOTHESES 473, 474-77 (2009).

⁴⁸² See Geddes & Talbert, *supra* note 383, at 627-29.

⁴⁸³ *Id.* at 629.

The only other literature support for this hypothesis stems from a 2010 case report by Barnes and colleagues.⁴⁸⁴ In that case, the authors report on a 4.5-month-old infant who was reportedly found by his father choking and blue.⁴⁸⁵ The infant ultimately died after transportation to the Emergency Department and a short PICU course.⁴⁸⁶ His findings included bilateral SDH, SAH, and acute rib fractures.⁴⁸⁷ While in the PICU, an eye examination revealed retinal hemorrhages throughout both eyes (no normal retina were identified in either eye) with 360° retinal detachment.⁴⁸⁸ The authors reported that the child had not suffered AHT and that “choking, vomiting, or paroxysmal coughing (e.g., pertussis) may also result in SDH and RH.”⁴⁸⁹ The authors concluded that the SDHs and RHs are “consistent with the history of infantile dysphagic choking as consistently provided by the caretaker.”⁴⁹⁰

With regards to neck injury, a recent paper by Matshes et al. set forth the hypothesis that hypoxia was indeed the underlying cause for SDHs and RHs, but that neck injury, as opposed to choking or coughing, was the preceding cause of the hypoxia.⁴⁹¹ In that paper, Matshes et al. reported on 35 non-sequential infant fatalities from 3 different medical examiners’ offices. Twelve (34%) of these were “confirmed or suspected by history and circumstance to have been subjected to hyperextension and hyperflexion forces.”⁴⁹² The authors did not describe how the infants were selected or how the neck injury was determined. These 12 hyperflexion/hyperextension cases were compared with 23 control infants in whom neck hyperextension/hyperflexion was not identified or suspected.⁴⁹³ The authors reported that all 12 hyperextension/hyperflexion cases had nerve root hemorrhage (bleeding where the nerves of the neck enter the spinal cord), while only 1 of the 23 control infants had nerve root hemorrhage.⁴⁹⁴ The authors indicated that the nerve root hemorrhage in the “shaking injury” infants was evidence that they sustained neck injuries that interrupted the regulation of breathing, resulting in

⁴⁸⁴ See Barnes et al., *supra* note 383, at 7, 9-10.

⁴⁸⁵ *Id.* at 7.

⁴⁸⁶ *Id.* at 7-8.

⁴⁸⁷ *Id.* at 8.

⁴⁸⁸ *Id.*

⁴⁸⁹ *Id.*

⁴⁹⁰ *Id.*

⁴⁹¹ See Matshes, *supra* note 307.

⁴⁹² *Id.*

⁴⁹³ *Id.*

⁴⁹⁴ *Id.*

hypoxia.⁴⁹⁵ They reported that all 12 “shaking injury” cases had SDH, while only 2 of the 23 control infants had SDH.⁴⁹⁶ This was a statistically significant relationship comparing SDH amongst those with shaking as compared with those without shaking (chi square, $p=0.000$). The authors concluded that the 12 “shaking injury” cases had neck injuries that resulted in hypoxia and a subsequent SDH.⁴⁹⁷

D. Scientific critique of the adjunct hypotheses

While Geddes and Talbert’s computer model is interesting and, perhaps, hypothesis generating, no clinical or physiological data exist to support it. Geddes and Talbert themselves admit that the values utilized in their model are calculations and have not been demonstrated to be true.⁴⁹⁸ The authors state that although their model demonstrates the pressure within the vessels surpasses the failure threshold, “[n]o research specifically addressing the question of stress failure of intracranial veins appears to have been reported in the literature.”⁴⁹⁹ Furthermore, since its publication six years ago, there has been *no* subsequent confirmation or supporting research published. Finally, given the ubiquity of infant gastroesophageal reflux (spitting up), if choking or gagging on formula were truly a meaningful cause of death, SDH, or RH, would it not already have been identified and published in medical treatises and literature? Yet, this is not the case.

With regards to the [Barnes et al.](#) case report, there are several ethical concerns with this case report that impact the scientific validity of the data presented. First, the “case report” bears striking similarities to a case (*Zavian Thomas v. State of Texas*)⁵⁰⁰ in which each of the co-authors were expert defense witnesses (a disclosure that was not made when published in the medical literature). Second, when confronted in the medical literature with the similarities between this “case report” and that case,⁵⁰¹ the authors chose not to clarify this issue.⁵⁰² Finally,

⁴⁹⁵ *Id.* at 84.

⁴⁹⁶ *Id.*

⁴⁹⁷ *Id.* at 88.

⁴⁹⁸ See Geddes, *supra* note 305, at 629.

⁴⁹⁹ *Id.* at 630.

⁵⁰⁰ *Thomas v. State of Texas*, 03-07-00646-CR, 2009 WL 1364348 at 1, (Tex. App.—Austin May 14, 2009).

⁵⁰¹ Christopher. S. Greeley, *Letter to the Editor*, 17 SEMINARS IN PEDIATRIC NEUROLOGY 275, 277 (2010).

⁵⁰² John Galaznik et al., *Reply to Greeley et al.*, 17 SEMINARS IN PEDIATRIC NEUROLOGY 279 (2010).

there is concern that the “case report” did not present complete clinical information when published in the medical literature.⁵⁰³

One condition Talbert himself highlighted as an example of how choking or coughing, in the face of hypoxia, could cause SDH and RH is pertussis (whooping cough).⁵⁰⁴ Pertussis is an airway infection by the bacteria *Bordetella pertussis* which produces a toxin that causes extensive lung inflammation.⁵⁰⁵ Prior to the advent of an effective vaccination for the bacteria, there were over 200,000 cases annually in the US, mostly in infants and children.⁵⁰⁶ Tens of thousands of deaths occurred each year in the US, with over three-quarters of deaths being children younger than two years.⁵⁰⁷

Despite the overwhelming burden of this disease, there have been only two cases of pertussis-associated SDH in over 100 years of published medical literature. The first, from 1885,⁵⁰⁸ was a two-year old who died from reported pertussis. The second case was reported in the American Academy of Pediatrics Red Book (infectious diseases manual).⁵⁰⁹ The latter involved a four-week old from 1968 who had pertussis and developed *Staph aureus* pneumonia.⁵¹⁰ The infant died as a complication of pneumonia brought on by pertussis.⁵¹¹

As with gastroesophageal reflux, given the extensive history of pertussis, with hundreds of thousands of deaths in infants and children, if SDH or RH were a notable feature, would they not already be identified and reported in the medical literature? Yet, this is also not the case. Furthermore, in a well-designed prospective study, conducted over a 4-year period, Curcoy et al. examined the eyes of 35 infants and young children admitted to their hospital with pertussis.⁵¹²

⁵⁰³ While the authors reported an acute rib fracture was from CPR, the vignette, as presented in the medical literature, did not mention the presence of a healing rib fracture. See Barnes, *supra* note 306, at 10. A review of the court transcripts indicates that at least one of the co-authors was clearly aware of it. Thomas, 2009 WL 1364348.

⁵⁰⁴ See Talbert, *supra* note 358.

⁵⁰⁵ DIVISION OF BACTERIAL DISEASES, NATIONAL CENTER FOR IMMUNIZATION AND RESPIRATORY DISEASES, *Pertussis: Disease Specifics* (2012), <http://www.cdc.gov/pertussis/clinical/disease-specifics.html>.

⁵⁰⁶ James D. Cherry, *Pertussis in the Preantibiotic and Prevaccine Era, with Emphasis on Adult Pertussis*, CLINICAL INFECTIOUS DISEASES (SUPPLEMENT), S107, S107 (1999).

⁵⁰⁷ *Id.* at S109.

⁵⁰⁸ J.N. Marshall, *Aphasia and Cerebral Haemorrhage Complicating Whooping-Cough*, 23 GLASGOW MED. J. 24 (1885). While this is often cited as a case with SDH, the manuscript actually indicates that there was not an SDH, as is often contended there was.

⁵⁰⁹ AMERICAN ACADEMY OF PEDIATRICS: *Pertussis*, in RED BOOK: 2003 REPORT OF THE COMMITTEE OF INFECTIOUS DISEASES 472 (Pickering ed., 2003).

⁵¹⁰ *Id.*

⁵¹¹ *Id.*

⁵¹² Ana I. Curcoy et al., *Is Pertussis in Infants a Potential Cause of Retinal Haemorrhages?*, 97 ARCH. DIS. CHILD. 239 (2012).

They found *none* with retinal hemorrhages. Similar findings were repeated in a prospective study by Goldman et al.⁵¹³ Finally, Herr et al. prospectively examined the eyes of 100 infants admitted to the Children's Hospital of Pittsburgh with forceful vomiting caused by pyloric stenosis.⁵¹⁴ They also found *none* with retinal hemorrhages. It appears that there *is* little scientific data to support the hypothesis that coughing or choking causes SDH or RH.

With regards to the hypothesis of neck injury as a putative cause of SDHs or RHs, beyond the small numbers in the Matshes study, this paper has some significant methodological flaws that make meaningful interpretation impossible. The authors were not blinded to the neck findings, clinical information, or the pathologic findings. How neck hyperextension/hyperflexion was determined was not described.⁵¹⁵ As neck hyperextension/hyperflexion was a key determinant in whether the infant was a case or control, it needed to be explained how it was actually determined. Finally, and very interestingly, 4 of the 12 (33%) cases of shaking injury did not have evidence of hypoxic encephalopathy yet still had SDH.⁵¹⁶ Clearly, Matshes' data more strongly supported shaking alone as the cause of SDH, rather than hypoxia.

E. Conclusion

From the clinical perspective, hypoxia is an important consideration. Hypoxia likely plays a role in some of the significant neuro-devastation seen in infants and children who are victims of any traumatic brain injury, not just AHT. However, when it comes to hypoxia causing SDH and RH, there simply is no clinical data or compelling research that supports this contention. *If examined on the Oxford Centre for Evidence-Based Medicine rating scale,*⁵¹⁷ *the current level of evidence on the topic is of the lower kind—level 4. Comparatively, the level of evidence arguing against this hypothesis is stronger—level 2b.*

⁵¹³ Michael Goldman et al., *Severe Cough and Retinal Hemorrhage in Infants and Young Children*, 148 J. PEDIATRICS 835, 836 (2006).

⁵¹⁴ Sandra Herr et al., *Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants with Vomiting Caused by Pyloric Stenosis*, 113 PEDIATRICS 1658 (2004).

⁵¹⁵ See generally Matshes, *supra* note 366. In fact, some cases were included if neck hyperflexion/hyperextension was merely suspected. *Id.* at 83.

⁵¹⁶ *Id.* at 87.

⁵¹⁷ See CENTRE FOR EVIDENCE BASED MEDICINE, UNIVERSITY OF OXFORD, LEVELS OF EVIDENCE (2009), available at www.cebm.net/index.aspx?o=1025.

THE DAUBERT ANALYSIS

The determination of what is “reliable” expert testimony is a problem that has vexed jurists and legal scholars for hundreds of years. In one of the earlier historical legal writings on the subject matter, one eminent jurist and legal scholar, Judge Learned Hand, wrote:

“Having briefly considered the history of the present position of expert witnesses, the really practical question is whether it is the best way to use the information they can give. *There are two things I wish to prove: first, that logically the expert is an anomaly; second, that from the legal anomaly serious practical difficulties arise.*”⁵¹⁸

Although Judge Hand offered his brethren of the long robe a solution to the predicament of the expert witness,⁵¹⁹ this legal malady continues to ail jurisprudence to this very day.

For scores, the common law rule governing admissibility of scientific expert testimony was the *Frye* standard (or the “general acceptance” test).⁵²⁰ With the enactment of the Federal Rules of Evidence (FRE) in 1975, and the *Daubert* court’s subsequent countenance of them in many jurisdictions, the *Frye* standard came to pass.⁵²¹ FRE 702 states that expert witness testimony shall be restricted to “scientific, technical, or other specialized knowledge,” that is the “product of reliable principles and methodology.”⁵²² The *Daubert* court interpreted the adjective “scientific” to mean “a grounding in the methods and procedures of science”⁵²³ and tethered “evidentiary reliability” to “scientific validity.”⁵²⁴ From there, the *Daubert* court enunciated its renowned checklist of factors for assessing “scientific validity.”⁵²⁵ Subsequent elaboration on the limits and meanings of FRE 702 in *Joiner*⁵²⁶ and *Kumho*⁵²⁷ have clarified that the expert’s

⁵¹⁸ Learned Hand, *Historical and Practical Considerations Regarding Expert Testimony*, 15 HARV. L. REV., 40, 50 (1901) (emphasis added).

⁵¹⁹ In his writings, Judge Hand argued for the existence of “competent tribunal” or “a single expert,” not called by either side, “who have possessed themselves the specialized experience” and “trained powers of observation,” to “advise the jury of the general propositions applicable to the case.” *See id.* at 55-56.

⁵²⁰ *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923).

⁵²¹ The *Frye* “general acceptance” test remains the rule governing admissibility of scientific expert testimony in several states, including California, Florida, Illinois, Kansas, Maryland, Minnesota, New Jersey, New York, Pennsylvania, and Washington. Alice Lustre, *Post-Daubert Standards of Admissibility of Scientific and Other Expert Evidence in State Courts*, 90 A.L.R.5th 453, §§28-43 (2001).

⁵²² FED. R. EVID. 702.

⁵²³ *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 590 (1993).

⁵²⁴ *Id.* at 590-591, n.9.

⁵²⁵ 1) Falsifiability; 2) Peer review and publication; 3) Known or potential rate of error; and, 4) General acceptance. *Id.* at 593-594.

⁵²⁶ *General Electric Co. v. Joiner*, 522 U.S. 136 (1997).

methodology cannot be based solely upon “the ipse dixit of the expert”⁵²⁸ and must be “properly applied” to the particular “facts of the case.”⁵²⁹

In child abuse cases, the gatekeeper is confronted with primarily⁵³⁰ three legal issues:

- 1) Does a physician’s testimony constitute “scientific,” “technical,” or “other specialized knowledge?”;
- 2) Is the physician’s testimony the “product of reliable principles and methodology?”; and,
- 3) Has the physician reliably applied those principles and methodology to the particular facts of the case?

The first and second issues are generally addressable here; the third requires specific application to specific cases with specific fact patterns.

A. Does a physician’s testimony in child abuse cases constitute “scientific,” “technical,” or “other specialized knowledge”?

Although not the dispositive issue, it is not a superfluous matter to determine whether a physician’s testimony in child abuse cases constitutes “scientific,” “technical,” or “other specialized knowledge.” Many legal scholars have noted that the adjective “scientific” connotes a greater reliability and even an “aura of infallibility.”⁵³¹ Although empiric data from civil and criminal juries *has* not supported that “aura of infallibility” concern, empiric data *has* confirmed increased levels of juror attention to, critique of, and reliance upon appropriately presented scientific information.⁵³²

One distinguished evidentiary scholar has asserted that physician testimony in support of shaken baby syndrome is “non-scientific,” stating that it is premised upon primarily “anecdotal”

⁵²⁷ *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999).

⁵²⁸ *See Joiner*, *supra* note 384, at 146.

⁵²⁹ *See Kumho*, *supra* note 385, at 157.

⁵³⁰ While the gatekeeper must also assess relevance and potential 403(b) objections, our analysis will focus on these three legal questions.

⁵³¹ *See United States v. Addison*, 498 F.2d 741, 744 (1974) (The Court stated, “scientific proof may in some instances assume a posture of mystic infallibility in the eyes of a jury of laymen.”). *See also* J.W. Strong, *Language and Logic in Expert Testimony: Limiting Expert Testimony by Restrictions of Function, Liability, and Form* (1992) 71 OR. L. REV. 349, 367, n.81 (“There is virtual unanimity among courts and commentators that evidence perceived by jurors to be ‘scientific’ in nature will have particularly persuasive effect”); Neil Vidmar & Shari S. Diamond, *Juries and Expert Evidence*. 66 BROOK. L. REV. 1121, 1125 (2000) (quoting an Amicus brief filed on behalf of the defendant in *Kumho Tire Co. Ltd. v. Carmichael*, 526 U.S. 137 (1999), “[Because of the ‘aura of infallibility’], ‘even when jurors have a ‘basis for questioning the expert’s reliability [they] may be disinclined to do so.’ ”).

⁵³² *See generally*, Vidmar & Diamond, *supra* note XXX, at 1140–49.

evidence.⁵³³ That learned scholar goes on to assert that physician testimony against shaken baby syndrome is “scientific,” arguing that it attains “empiric” validity from Duhaime’s biomechanical study in 1987.⁵³⁴ Although the learned scholar makes no clarification as to what constitutes “anecdotal” evidence or whether there is evidence other than “anecdotal” evidence in support of shaken baby syndrome, he still concedes its admissibility under *Daubert* scrutiny.⁵³⁵ The shortcomings of this “scientific”/“non-scientific” analysis will be discussed in further detail below.

In defining “scientific,” the *Daubert* court stated that “the adjective ‘scientific’ implies a grounding in the methods and procedures of science.”⁵³⁶ Generally speaking, a physician’s testimony has been and is considered “scientific.” A physician’s education includes the scientific basis of health and disease.⁵³⁷ Rooted in a foundation of core science subjects, such as biology, physics, chemistry, and biochemistry, physicians are trained to apply those scientific principles to the human body to better understand the physiology of the body and the pathology of disease that can affect it. Physicians are further trained to use scientific literature to compare alternative approaches to diagnosis and treatment.⁵³⁸ In addition, physicians receive basic training on statistical analysis, often applying those principles to critically evaluate the medical literature.

Courts that have confronted the issue have commented, “[c]linical diagnoses bear the marks of science.”⁵³⁹ As the American Medical Association stated in its amicus brief in *Daubert*:

‘Scientific knowledge’ is that body of knowledge that has been learned or developed in accordance with rigorous scientific methodology. The scientific method involves replicable, empirical testing of hypotheses.... *Medical knowledge is one kind of scientific knowledge.* It is

⁵³³ Edward Imwinkelried, *Shaken Baby Syndrome: A Genuine Battle of the Scientific (and Non-Scientific) Experts*, 46 CRIM. L. BULL. 156, 186–87 (2010) (The author asserts that proponents of the syndrome primarily rest their support upon the “admissions by caretakers” in Caffey’s 1974 article and on the “experience” of “forensic pathologists.”).

⁵³⁴ *Id.* at 184.

⁵³⁵ *Id.* at 188.

⁵³⁶ *Daubert*, *supra* note XXX, at 590.

⁵³⁷ William Stead & John Starmer, *Beyond Expert-Based Practice*, in ., EVIDENCE-BASED MEDICINE AND THE CHANGING NATURE OF HEALTH CARE: 2007 IOM ANNUAL MEETING SUMMARY 94 (INST. OF MED., Mark McClellan et al. eds, 2008).

⁵³⁸ *Id.*

⁵³⁹ See *State v. Sanchez-Cruz*, 33 P.3d. 1037, 1042 (Or. App. 2001) (emphasis in original) (citing *Jennings v. Baxter Healthcare Corp.*, 331 Or. 285, 299–301, 14 P.3d 596 (2000)).).

acquired through the application of the scientific method to questions concerning the effects of various interventions on human health.⁵⁴⁰

In fact, after considering whether the expert testimony of the physicians and biostatisticians involved in the case was “scientific, technical or other specialized knowledge,” the *Daubert* court concluded that their analysis was “limited to the *scientific context because that is the nature of the expertise offered here*.”⁵⁴¹

Additionally, some courts and scholars have made a distinction between “hard” and “soft” sciences.⁵⁴² Because this distinction has no true correlation with reliability, we will not delve into further discussion of this issue.

The natural extension of this analysis is to consider whether there are any features or characteristics of a physician’s methodology in AHT/SBS cases that make it less scientific or non-scientific. The simple answer is no. The physician in AHT/SBS cases employs no different methodology than the ER physician who assesses life or death scenarios in the emergency room, or than the neurosurgeon who assesses the cause and treatment of intracranial bleeds, or than the forensic pathologist who assesses the cause and manner of death in a variety of cases. All employ the “differential diagnosis” methodology, a methodology rooted in the scientific method.⁵⁴³

Furthermore, if, as the *Daubert* court stated, the “scientific method” is a “process” of “generating hypotheses and testing them to see if they can be falsified,”⁵⁴⁴ then it is without question that AHT/SBS has been subjected to the “scientific method.” As Narang pointed out in his prior article, physicians from all across the world have not only tested AHT/SBS from a variety of different perspectives but in a variety of different disciplines: biomechanics, pathology, radiology, ophthalmology, neurosurgery, and general pediatrics.⁵⁴⁵ In its recent assessment of the forensic sciences, the National Research Council stated:

⁵⁴⁰ Brief of the American Medical Association et al., as Amici Curiae Supporting Respondents at 4, *Daubert v. Merrel Dow Pharmaceuticals, Inc.*, 951 F.2d 1128 (9th Cir. 1991) (No. 90-55397), 1993 WL 13006285 (emphasis added).

⁵⁴¹ See *Daubert*, *supra* note 381, at 590, n.8 (emphasis added).

⁵⁴² See *State v. McMullen*, 900 A.2d 103, 114-15 (Del. Super. Ct. 2006).

⁵⁴³ See *id.* at 116-19; see generally McClellan et al., *supra* note XXX, at 94. This is to be distinguished from “differential etiology,” which is a “legal invention not used by physicians.” JOHN B. WONG ET AL., FED JUDICIAL CTR., *Reference Guide on Medical Testimony*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 691 (3rd ed. 2011) available at http://books.nap.edu/openbook.php?record_id=13163&page=R1.

⁵⁴⁴ See *Daubert*, *supra* note 381, at 590, 593.

⁵⁴⁵ Narang, *supra* note 10, at 539-540, 583.

Scientists continually observe, test, and modify the body of knowledge. Rather than claiming absolute truth, science approaches truth either through breakthrough discoveries or incrementally, by testing theories repeatedly.⁵⁴⁶

In fact, it is this very scientific methodology—this process for seeking greater scientific precision—that has prompted physicians to modify constrictive terminology, such as “shaken baby syndrome,” to more inclusive terminology, such as “abusive head trauma.” However, as child abuse physicians have utilized the science to be more precise, they have ironically been criticized for being “less scientific.”⁵⁴⁷

Finally, the assertion that the evidence basis in support of AHT/SBS is “primarily anecdotal” is not only factually inaccurate, but logically overly simplistic. “Anecdotal evidence” refers to evidence from anecdote, or more simply speaking, evidence that is primarily based upon personal experience and not subjected to the rigors of scientific analysis and scrutiny.⁵⁴⁸ While certainly some evidence of shaking has been “anecdotal” (*i.e.*, some admissions of shaking by caretakers to physicians), there are other forms of scientific evidence that support the “shaking” proposition. Recently, animal studies (on species that have highly similar head and neck anatomic structures to the human infant) have reproduced the very injuries—SDHs and RHs—reported to be found as a result of shaking.⁵⁴⁹ And as detailed above, there are biomechanical studies that support the “shaking” proposition.⁵⁵⁰ More importantly, shaking accounts of perpetrators have not been merely anecdotally asserted and accepted, but have been subjected to various aspects of scientific scrutiny, from examination of the specifics and repetitiveness of

⁵⁴⁶ See COMMITTEE ON IDENTIFYING THE NEEDS OF THE FORENSIC SCIENCES COMMUNITY, NAT’L RESEARCH COUNCIL, *Strengthening Forensic Science in the United States: A Path Forward* 112 (2009), available at <http://www.nap.edu/catalog/12589.html>.

⁵⁴⁷ See Findley et al., *supra* note 3, at 218-220.

⁵⁴⁸ *Anecdotal Definition*, MERRIAM-WEBSTER.COM, <http://www.merriam-webster.com/dictionary/anecdotal> (defining “anecdotal evidence” as “based on or consisting of reports or observations of usually unscientific observers”). See DAVID H. KAYE & DAVID A. FREEDMAN, FED. JUDICIAL CTR., *Reference Guide on Statistics*, in REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 90-92 (2nd ed. 2000).

⁵⁴⁹ See Finnie et al., *supra* note 242.

⁵⁵⁰ See *supra* Part V, Biomechanics.

shaking events⁵⁵¹ to the correlation of those accounts to the presence or absence of physical trauma signs upon the body.⁵⁵²

However, and most importantly, the evidence for or against “shaking” is not merely a biomechanical question. The analysis does not simply end at the question of whether the estimated forces required to cause SDHs have or have not been reproduced in biomechanical studies; there are clinical questions as well. Assuming, *arguendo*, that impact with a surface (soft or hard) is required to reach the estimated force thresholds of SDHs, then what? Because it is already well-established that many abusive head injuries do occur with impact against a soft surface, what next? If valid probabilistic judgments are to be made, further *clinical* questions must be answered—such as, with what frequency/commonality do the associative findings (SDHs, RHs, fractures, etc.) occur? What degree of reliability is there in those findings? How common are those findings in low impact/simple short fall events? And are there other clinical variables that are strongly indicative of accidental or abusive events? The focus on solely the biomechanical question is itself a prime example of anchoring bias.⁵⁵³ It is the assimilation and assessment of *all* types of evidence (as will be discussed in further detail herein below), not just biomechanical data, which leads to the most reasonable conclusion on this matter.

Therefore, physician testimony in AHT/SBS cases is, and should be, considered “scientific.”

B. Is the physician’s testimony in AHT/SBS cases the “product of reliable principles and methodology”?

As mentioned above, determining what is “reliable methodology” in an expertise or a subject matter that is completely foreign to one’s own, is no simple task for anyone. Although the *Daubert* court listed a checklist of factors to consider in assessing “scientific reliability,”

⁵⁵¹ Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 PEDIATRICS 553-54. (2010).

⁵⁵² Suzanne P. Starling et al., *Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, 158 ARCHIVES PEDIATRIC ADOLESCENCE MED. 457 (2004); Erica Bell et al., *Abusive Head Trauma: A Perpetrator Confesses*, 35 CHILD ABUSE & NEGLECT 74-77 (2011).

⁵⁵³ *Anchoring Bias in Decision-Making*, SCIENCEDAILY.COM, <http://www.sciencedaily.com/articles/a/anchoring.htm>. “During normal decision making, individuals anchor, or overly rely, on specific information or a specific value and then adjust to that value to account for other elements of the circumstance.”

some courts have questioned the hard-and-fast application of those factors to clinical medicine.⁵⁵⁴ While not a perfect fit to clinical medicine, the *Daubert* factors are malleable to clinical medicine and help in the overall analysis of reliability.

As the *Daubert* court ultimately stated, it is the “*principles and methodology*,” not the “conclusions they generate,” that are of paramount importance.⁵⁵⁵ As Narang pointed out in his first part of this analysis, the methodology physicians employ in coming to the diagnosis of AHT is no different than the methodology physicians employ in arriving at *any* medical diagnosis—it is the differential diagnosis methodology.⁵⁵⁶

Legal scholars have expounded that, while the derivation of scientific principles involves inductive reasoning, in court, the explanation of the methodology follows a deductive, syllogistic format:

Although scientific propositions are derived inductively, in the courtroom scientific testimony is ordinarily presented in a deductive, syllogistic format.... *The major premise is a principle, procedure, or explanatory theory derived by the inductive, scientific technique.* The physician applies that major premise to the facts of the case, namely, plaintiff's case history. *The symptoms displayed by this specific plaintiff are the witness' minor premise.* That case history might show that plaintiff has experienced symptoms A, B, and C. *The result of applying the major to the minor premise is a conclusion, the witness' opinion on the merits of the case....* Hence, the “path to the witness' final opinion” leads through the major and minor premises on which the expert relies.⁵⁵⁷

Applying such to AHT/SBS cases, the “major premise” would be the scientific principles and evidence underlying the AHT/SBS diagnosis. The “minor premise” would be the utilization of the differential diagnosis methodology to the specific facts of the AHT/SBS case.

Thus, the analytic journey from here courses through three paths: 1) determining whether the scientific principles and evidence underlying the AHT/SBS diagnosis are in fact reliable, i.e.,

⁵⁵⁴ See *McMullen*, *supra* note 378, at 114 (stating, “Because the objectives, functions, subject matter and methodology, of hard science vary significantly from those of the discipline of clinical medicine, as distinguished from research or laboratory medicine, the hard science techniques or methods that became the ‘*Daubert* factors’ generally are not appropriate for assessing the evidentiary reliability of a proffer of expert clinical medical testimony.”)

⁵⁵⁵ See *Daubert*, *supra* note 381, at 594-95. (emphasis added).

⁵⁵⁶ See Narang, *supra* note 10, at 583-84.

⁵⁵⁷ Edward Imwinkelried, *The “Bases” of Expert Testimony: The Syllogistic Structure of Scientific Testimony*, 67 N.C. L. REV. 1, 2-3 (1988) (emphasis added); see also Hand, *supra* note 376, at 51-52 (describing expert testimony as the application of the “major premise” to the “minor premise”).

the “major premise”; 2) defining what exactly the “differential diagnosis methodology” is; and, 3) exploring whether the “differential diagnosis methodology” is, in general, a reliable methodology for applying the major premise to the specific facts of a case, i.e., the “minor premise.” The determination of whether a physician has validly applied the differential diagnosis methodology to a specific fact pattern is a mental endeavor a particular gatekeeper must endure. Toward the conclusion of this article, we will discuss some tools available to the gatekeeper for confronting this challenge.

1) Are the scientific principles and evidence underlying the AHT/SBS diagnosis “reliable” (i.e., the “major premise”)?

The Honorable Justice Stephen Breyer commented:

The search is not a search for scientific precision. We cannot hope to investigate all the subtleties that characterize good scientific work. A judge is not a scientist, and a courtroom is not a scientific laboratory. But consider the remark made by the physicist Wolfgang Pauli. After a colleague asked whether a certain scientific paper was wrong, Pauli replied, “That paper isn’t even good enough to be wrong!” Our objective is to avoid legal decisions that reflect that paper’s so-called science. The law must seek decisions that fall within the boundaries of scientifically sound knowledge.⁵⁵⁸

Justice Breyer’s statements are not merely colorful commentary or interesting narrative. They express the careful balance that is sought between two competing evidentiary goals—scientific soundness and a liberal, flexible approach to admissibility. They are notable in light of recent court decisions employing stricter standards of admissibility⁵⁵⁹ and judicial surveys expressing confusion regarding the proper criteria for admissibility.⁵⁶⁰ And they are especially

⁵⁵⁸ See Breyer, *supra* note 8, at 4.

⁵⁵⁹ See *Hamilton*, *supra* note 4; Imwinkelried, *supra* note 399 at 14 n.105 (quoting *State v. Hyatt*, No. O6MJ-CR00016-02 (Mo.Cir.Ct. Nov. 6, 2007). “[I]n an unpublished order, the trial judge found that the prosecution had not met its burden of proving that shaken baby syndrome is generally accepted in the scientific and medical circles”).

⁵⁶⁰ Sophia Gatowski et al., *Asking the Gatekeepers: A National Survey of Judges on Judging Expert Evidence in a Post-Daubert World*, 25 LAW & HUMAN BEHAVIOR 433, 444-47 (2001).

noteworthy because some legal scholars have fallaciously confounded standards for diagnostic sufficiency with standards for criminal conviction sufficiency.⁵⁶¹

So the question is simple: Is the science underlying AHT/SBS “junk science,” or science “that’s not even good enough to be wrong?” One legal scholar made the following assertions that formulated the premise of Narang’s first article:

- 1) that “the scientific underpinnings of SBS have crumbled over the past decade”⁵⁶²;
- 2) that, “as evidence-based medicine...required doctors to derive their research from methods that are scientific and statistically rigorous,” doctors learned that the diagnosis was predicated upon “flawed science”⁵⁶³; and
- 3) that “as technology and scientific methodology advanced, researchers questioning the basis for SBS reached a critical mass”⁵⁶⁴ (i.e., no longer a “general acceptance”).

These assertions had been “reified” in prior⁵⁶⁵ and subsequent legal commentary,⁵⁶⁶ and unfortunately, found scientifically un-scrutinizing ears in three Supreme Court justices in the recent *Cavazos v. Smith* decision.⁵⁶⁷ Simply stated, these assertions question the reliability of the scientific principles that formulate the physician’s “major premise” in the syllogistic argument.

In response to those assertions, in the first part of this analysis, Narang examined the scientific underpinnings of two findings commonly seen in AHT/SBS—SDHs and RHs. Narang examined the science supporting the association of these findings with AHT/SBS. As evidence of the reliability of those associative findings, Narang referenced over 200 evidence-based, scientific studies (not editorials or reviews—but *clinical studies*), detailed the scientific validity of 15 of those articles, and subjected them to the 4 *Daubert* factors used in assessing scientific

⁵⁶¹ See Findley et al., *supra* note 3, at 286-87 (in discussing the scientific literature, the authors state, “[e]ven if the causes were accurately classified, however, this measure [the p-value] provides no indication of the *strength* of the correlation for it does not distinguish between weak correlations...and strong ones...*Yet the strength of the correlation is precisely what is needed to satisfy fact finding requirements in criminal cases, which requires proof beyond a reasonable doubt. Statistical significance is necessary but not sufficient to support this evidentiary standard.*”) (emphasis added) .

⁵⁶² See Tuerkheimer, *supra* note 3, at 11.

⁵⁶³ *Id.* at 12.

⁵⁶⁴ *Id.* at 14.

⁵⁶⁵ See Molly Gena, *Shaken Baby Syndrome: Medical Uncertainty Casts Doubt on Convictions*, 3 WISC L. REV. 701, 701-727 (2007); Matthew D. Ramsey, *A Nuts and Bolts Approach to Litigating the Shaken Baby or Shaken Impact Syndrome*, 188 MILITARY L. REV. 1, 1 (2006).

⁵⁶⁶ See Findley et al., *supra* note 3; Imwinkelried, *supra* note 391.

⁵⁶⁷ See *Cavazos*, 565 U.S. __ (2011)..

reliability.⁵⁶⁸ In conclusion, Narang determined that not only did this scientific literature meet *Daubert* criteria for reliability but that there was no “critical mass” questioning the diagnosis or change in general acceptance of the diagnosis.⁵⁶⁹

Interestingly, having now been confronted with promulgation of that scientific literature, those same scholars have shifted their arguments.⁵⁷⁰ Whereas before they argued that there was little-to-no evidence-based medical literature supporting the diagnosis (likening it to an “inverted pyramid with a small database”),⁵⁷¹ they have now posited that Narang’s use of the “voluminous” scientific literature “serves to intimidate those who are not familiar with its methodological shortcomings.”⁵⁷² Whereas before they alleged that the AHT/SBS literature did not engage in the scientific or statistical rigors required by evidence-based medicine,⁵⁷³ now they state that the literature either misstates the significance of the *P-value* or does not sufficiently calculate the strength of statistical associations by providing posterior probabilities.⁵⁷⁴ And whereas before they asserted that “researchers questioning the basis for SBS reached a critical mass,”⁵⁷⁵ now they argue either that “it is increasingly difficult to gauge the extent to which doctors in general agree”⁵⁷⁶ or that it is “insufficient to rely on the fact that some professional groups accept or endorse the diagnosis of SBS/AHT.”⁵⁷⁷

While it certainly would be entertaining to engage in ongoing debate about “prosecutor’s fallacies,” “improper classifications,” and “shifting paradigms,” such an endeavor would be neither productive nor relevant. Ultimately, at this point in the analysis, the focus must be on whether the scientific principles forming the basis of the AHT diagnosis are reliable, i.e., whether there is reliability in the physician’s major premise of the syllogistic argument. It is important to note at this point that it is simply beyond the scope of this article to state *ALL* the scientific principles a physician utilizes in arriving at the AHT/SBS diagnosis. For example,

⁵⁶⁸ See Narang, *supra* note 10, at 596-627.

⁵⁶⁹ See generally, *Id.* at 541-60.

⁵⁷⁰ *Id.* at 576-88.

⁵⁷¹ See Tuerkheimer, *supra* note 3, at 12-13 n.79 (quoting Patrick D. Barnes, *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, 18 TOPICS MAGNETIC RESONANCE IMAGING 53, 55 (2007).

⁵⁷² See Findley et al., *supra* note 3, at 296.

⁵⁷³ See Tuerkheimer, *supra* note 3, at 12-13.

⁵⁷⁴ See Findley et al., *supra* note 3, at 286-88.

⁵⁷⁵ See Tuerkheimer, *supra* note 3, at 14.

⁵⁷⁶ See Findley et al., *supra* note 3, at 242.

⁵⁷⁷ *Id.* at 289.

there are many developmental principles of infants and children that physicians utilize in correlation with a history in assessing the reliability of that history. Additionally, there are principles derived from various studies investigating alternative causes of SDHs, RHs, fractures, and bruises that physicians utilize in determining whether to rule out other potential causes of those findings.⁵⁷⁸

In his first article, Dr. Narang attempted to highlight *some* of the key scientific principles regarding SDHs and RHs. However, despite repetition,⁵⁷⁹ this still resulted in confusion by some scholars⁵⁸⁰ about what exactly those scientific principles were. Thus, given the importance of those principles to the overall analysis, we will restate those scientific principles below (along with their evidence-based medicine levels) in bold, so as to hopefully avoid any further confusion:

- 1) That trauma is the most common cause of SDHs—based upon epidemiologic studies in young children, both prospective and retrospective, from multiple countries (*level 3b evidence*);⁵⁸¹**
- 2) That when examining the breakdown of trauma-caused SDHs (i.e., accidental v. non-accidental), non-accidental trauma is by far more common—based upon epidemiology and pathology studies in young children, both prospective and retrospective (*level 2b evidence*);⁵⁸²**

⁵⁷⁸ Rebecca Crosier, *Shaken Baby Syndrome*, SHAKEN BABY SYNDROME, <http://neurowiki2012.wikispaces.com/Shaken+Baby+Syndrome>. (discussing in Part 2.2 and 2.3 alternative causes of SDH and RH and the need for medical professionals to be careful in a diagnosis of SBS.)

⁵⁷⁹ See Narang, *supra* note XX, at 548, 559, 571, 579, 595.

⁵⁸⁰ See Findley et al., *supra* note X, at 303 (“[i]n arguing admissibility under *Daubert*, moreover, it is unclear what Dr. Narang believes should be admitted. Evidence that some brain injuries in children are of traumatic origin, sometimes even intentionally inflicted? Evidence that subdural hematomas and retinal hemorrhages are seen in cases of inflicted abuse? Evidence that shaking can cause the triad and can lead to injury or death? Evidence that subdural hematomas and retinal hemorrhages are diagnostic of shaking or abuse in the absence of a major motor vehicle accident, fall from a multistory building or other proven alternative? Some of these questions are not controversial ...”).

⁵⁸¹ C. Hobbs et al., *Subdural Haematoma and Effusion in Infancy: An Epidemiological Study*, 90 ARCHIVES DISEASE CHILDHOOD 952, 954 (2005); Victoria Trenchs et al., *Subdural Haematomas and Physical Abuse in the First Two Years of Life*, 43 PEDIATRIC NEUROSURGERY 352, 352–53, 356 (2007); Dimitra Tzioumi & R. Kim Oates, *Subdural Hematomas in Children Under 2 Years. Accidental or Inflicted? A 10-Year Experience*, 22 CHILD ABUSE & NEGLECT 1105, 1106–07 (1998).

⁵⁸² Kenneth W. Feldman et al., *The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study*, 108 PEDIATRICS 636, 638 (2001) (finding that 59% of trauma SDHs were “intentional”, but only 23% were “accidental”); Jakob Matschke et al., *Nonaccidental Head Injury is the Most Common Cause of Subdural Bleeding in Infants < 1 Year of Age*, 124 PEDIATRICS 1587, 1594 (2009) (finding that 93% of trauma SDHs were “non-

- 3) That SDHs being much more common in non-accidental trauma than in accidental trauma is a statistically significant conclusion reached by numerous well-designed, prospective clinical studies (*level 2b evidence*);⁵⁸³
- 4) That severe RHs being much more common in non-accidental trauma than in accidental trauma is a statistically significant conclusion reached by numerous well-designed, prospective clinical studies (*level 2b evidence*);⁵⁸⁴
- 5) That severe RHs carry a high specificity and positive predictive value for non-accidental trauma—based upon prospective, validating clinical studies and systematic reviews (*level 1b and 2a evidence*);⁵⁸⁵
- 6) That the absence of a trauma history, in the presence of traumatic injuries, holds a high specificity and positive predictive value for non-accidental trauma—based upon several well-designed, prospective clinical studies (*level 2b evidence*).⁵⁸⁶

As Narang detailed in his first article, these scientific principles are the result of methodologies specifically designed to minimize circularity and bias.⁵⁸⁷ They produce results that have been repeatedly reproduced by other physicians.⁵⁸⁸ And they satisfy *Daubert* criteria for reliability.⁵⁸⁹

accidental” and only 7% were “accidental”); Hobbs et al., *supra* note X at 953 (finding that 94% of trauma SDHs were “non-accidental” and only 6% were “accidental”).

⁵⁸³ Ann-Christine Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger than 2 Years of Age*, 90 PEDIATRICS 179, 183 (1992); Kirsten Bechtel, et al., *Characteristics that Distinguish Accidental from Abusive Injury in Hospitalized Young Children with Head Trauma*, 114 PEDIATRICS 165, 165, 168 (2004); Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases*, 26 CHILD’S NERVOUS SYSTEM. 637, 638–39 (2010); Kent P. Hymel et al., *Mechanisms, Clinical Presentations, Injuries, and Outcomes from Inflicted Versus Noninflicted Head Trauma during Infancy: Results of a Prospective, Multicentered, Comparative Study*, 119 PEDIATRICS 922, 922 (2007); K. Hymel et al., *Head Injury Depth as an Indicator of Causes and Mechanisms*, 125 PEDIATRICS 712, 715–18 (2010).

⁵⁸⁴ *Id.*

⁵⁸⁵ See Vinchon, *supra* note XXX; Matthieu Vinchon et al., *Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study*, 102 J. NEUROSURGERY: PEDIATRICS 380, 380–81 (2005); Hymel et al., *supra* note XXX; S. Maguire, *Which Clinical Features Distinguish Inflicted from Non-Inflicted Brain Injury? A Systematic Review*, 94 ARCHIVES DISEASE CHILDHOOD 860, 860 (2009); Gaurav Bhardwaj et al., *A Systematic Review of the Diagnostic Accuracy of Ocular Signs in Pediatric Abusive Head Trauma*, 117 OPHTHAMOLOGY 983, 987 (2010); Shruti Agrawal et al., *Prevalence of retinal hemorrhages in critically ill children*, 6 PEDIATRICS 129 e1388-96 (2012).

⁵⁸⁶ Joeli Hettler & David S. Greenes, *Can the Initial History Predict Whether a Child with a Head Injury has been Abused?*, 111 PEDIATRICS 602, 602 (2003); Duhaime et al., *supra* note 425 at XX; Heather T. Keenan et al., *Child Outcomes and Family Characteristics 1 Year After Severe Inflicted or Noninflicted Traumatic Brain Injury*, 117 PEDIATRICS 317, 317 (2006).

⁵⁸⁷ See Narang, *supra* note 10, at 541-61.

⁵⁸⁸ See *id.*

⁵⁸⁹ See *id.*

Findley et al. responded by criticizing the validity of the literature on essentially two grounds— (1) that the methodology of “virtually all” of the studies is marred by “circularity”,⁵⁹⁰ and (2) that there are various errors clinicians have made in interpreting the results of these studies.⁵⁹¹ As “circular” methodology more directly pertains to the major premise (i.e., the scientific principles), it will be discussed below. As “interpretive errors” pertain more to the minor premise (i.e., the application of those principles to the particular facts of a case), they will be addressed in the “minor premise” section below.

Since “circularity” has been alleged to infect “virtually all” of the literature, prior to addressing it, it is appropriate to identify the scientific principles that are deducible from the Accidents, Bleeding Disorders, Biomechanics, and Hypoxia sections discussed above. These scientific conclusions are:

- 1) That short falls occurring in objective settings, such as hospitals, have not resulted in subdural hematoma or death—based upon several consecutive case series (*level 3b evidence*);⁵⁹²**
- 2) That severe injuries or death resulting from short falls are *rare* events—based upon well-designed, prospective studies and systematic reviews (*level 2a evidence*);⁵⁹³**
- 3) That certain clinical variables, such as apnea and severe RHs, demonstrate high positive predictive values for non-accidental traumabased upon prospective, validating clinical studies and systematic reviews (*level 1b and 2a evidence*);⁵⁹⁴**
- 4) That most bleeding disorders are rare, the more common bleeding disorders typically are mild, and intracranial hemorrhage resulting from bleeding disorders is a *rare* complication of the more severe rarer diseases—based upon clinical studies (*level 3b evidence; level 1b symptom prevalence evidence*);⁵⁹⁵**
- 5) That biomechanical studies have shown mixed results as to whether shaking can result in the estimated mechanical forces needed to cause SDHs;⁵⁹⁶**

⁵⁹⁰ See Findley et al., *supra* note X, at 274-75.

⁵⁹¹ *Id.* at 286-90.

⁵⁹² See *supra* Part 3.

⁵⁹³ *Id.*

⁵⁹⁴ *Id.*

⁵⁹⁵ See *supra* Part 4.

⁵⁹⁶ See *supra* Part 5.

- 6) That biomechanical studies have shown that RHs can result from shaking;⁵⁹⁷
- 7) That biomechanical studies have NOT shown that neck “failure” must result prior to the estimated forces required for SDHs being achieved;⁵⁹⁸
- 8) That macroscopic SDHs are not associated with hypoxia—based upon several well-designed radiology and pathology studies (*level 2b evidence*);⁵⁹⁹
- 9) That severe RHs are not associated with hypoxia—based upon well-designed clinical studies and animal studies (*level 2b evidence*);⁶⁰⁰
- 10) That adjunct hypotheses of hypoxia (such as “dysphagia/choking,” “coughing,” or “dural immature vascular plexus”) resulting in SDHs and/or RHs are supported by the lowest levels of evidence-based medicine (*level 4 or 5*), whereas evidence against such hypotheses is much stronger (*level 2b*).⁶⁰¹

As with the principles Narang discussed in his first article, and as demonstrated in the sections above, these scientific principles have been subjected to “falsifiability.” They have been peerreviewed and published. They represent the highest levels of statistical analysis. Their results have been reproduced in multiple studies and across various lines of research. And finally, as these principles form the basis of the generally accepted diagnosis of AHT/SBS,⁶⁰² they are generally accepted. Consequently, they are also valid and surpass *Daubert* scrutiny.

⁵⁹⁷ *Id.*

⁵⁹⁸ *Id.*

⁵⁹⁹ See *supra* Part 6; see also Narang, *supra* note 10, at 563–68. But see Irene Scheimberg et al., *Non-Traumatic Intracranial and Subdural Hemorrhage and Hypoxic Ischaemic Encephalopathy in Fetuses, Infants and Children Up to 3 Years of Age. Analysis of Two Audits of 636 Cases From Two Referral Centers in the UK*, PEDIATRIC & DEV. PATHOLOGY (2012) [Epub ahead of print].

⁶⁰⁰ See *supra* Part 6; Ana Isabel Curcoy et al., *Retinal Hemorrhages and Apparent Life-Threatening Events*, 26 PEDIATRIC EMERGENCY CARE 118 (2010); Raymond D. Pitetti et al., *Prevalence of Retinal Hemorrhages and Child Abuse in Children Who Present With an Apparent Life-Threatening Event*, 110 PEDIATRICS 557 (2002); Amy Odom et al., *Prevalence of Retinal Hemorrhages in Pediatric Patients After In-Hospital Cardiopulmonary Resuscitation: A Prospective Study*, 99 PEDIATRICS e3 (1997); Charanjit Kaur et al., *Early Response of Neurons and Glial Cells to Hypoxia in the Retina*, 47 INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCI. 1126 (2006); Taiji Nagaoka et al., *The Effect of Nitric Oxide on Retinal Blood Flow During Hypoxia in Cats*, 43 INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCI. 3037 (2002).

⁶⁰¹ See *supra* Part 6; see also Narang, *supra* note 10, at 505, 588–89; Michael Goldman et al., *Severe Cough and Retinal Hemorrhage in Infants and Young Children*, 148 J. PEDIATRICS 835 (2006); Sandra Herr et al., *Does Valsalva Retinopathy Occur in Infants? An Initial Investigation in Infants With Vomiting Caused by Pyloric Stenosis*, 113 PEDIATRICS 1658 (2004).

⁶⁰² Narang, *supra* note 10, at 574–76.

With regards to the “circularity” concerns raised by Findley et al.,⁶⁰³ interestingly, much like some of the other positions, this “circularity” argument has also shifted. Whereas before Findley et al. argued that the “circularity” consisted of including SDHs and/or RHs in the defining inclusion criteria of a study,⁶⁰⁴ now they argue that “major trauma” is the assumed circular premise in “virtually all” of the literature.⁶⁰⁵

Narang addressed this “circularity”⁶⁰⁶ argument in his first article.⁶⁰⁷ His retorts—of why “circularity” does not explain the historical articles (articles that initially identified these associative injuries prior to the designation of a syndrome) or why NO scientific studies have consequently been created that are not circular and show a lack of this association—have yet to be addressed. Setting aside these unrequited responses, there are several other fatal shortcomings to this argument.

First, as scientists, physicians are always asking how to test a hypothesis soundly. Well before the admonishments of Findley et al., physicians themselves recognized the methodological problems of circularity in some of the literature.⁶⁰⁸ In efforts to remedy these flaws, physicians created various *a priori* definitions for AHT (that excluded SDHs and RHs)⁶⁰⁹ and even limited some studies to simple comparative cohorts of witnessed accidents versus judicially confessed abusive acts.⁶¹⁰ Yet, Findley et al. *still* find these to be “circular.” If that is the case, then the simple question is: *what would be “non-circular methodology?”* If confessions are circular or invalid and all *a priori* definitions presume something, then what

⁶⁰³ See Findley et al., *supra* note 3, at 274. The critics have also lodged complaints of “observer bias” and “interpretive errors.” However, these relate more to the “minor premise” and thus will be discussed in further detail below.

⁶⁰⁴ See Tuerkheimer, *supra* note 3, at 13.

⁶⁰⁵ See Findley et al., *supra* note 3, at 274. .

⁶⁰⁶ “Circularity” is the logical fallacy in which the manner of proposing a question presumes an answer. For example, assume that scientists wished to prove that a pro sports team can win more games by hiring athletes who have, themselves, won many games. Using a single season’s data they compute the number of times each athlete has won or lost; they then compare these results to the number of times each team has won or lost. The design is clearly invalid—when a team wins, all its players win as well. The predictor variable (number of games the athlete has won) is simply a proxy for the variable being predicted. Consequently, a circular study tends to overstate the strength of an association.

⁶⁰⁷ See Narang, *supra* note 10, at 561–62.

⁶⁰⁸ See Am. Acad. of Pediatrics, *Inflicted Childhood Neurotrauma: Proceedings of a Conference Sponsored by Department of Health and Human Services, National Institute of Health, National Institute of Child Health and Human Development, Office of Rare Disease, and National Center for Medical Rehabilitation Research* (Robert M. Reece & Carol E. Nicholson eds., 2003).

⁶⁰⁹ See Duhaime, *supra* note 442; Hymel, *supra* note 442.

⁶¹⁰ See Vinchon, *supra* note 442.

would be a satisfactory methodology for a study? What is the sound methodology Findley et al. would utilize in conducting a study? This is a simple question that Findley et al. have yet to answer. It appears that what is guised as a “circular” critique is actually a philosophical and logical quagmire that devolves into the answer that “nothing,” then, could be non-circular.

Second, do doctors really *assume* that SDHs are caused by major trauma? Or is that not a *proven* entity? Closer scrutiny of the “circularity” label reveals that the study methodologies are not actually “circular” but simply *systematically deductive*. Narang spent considerable time in his first article detailing the historical progression of the understanding of SDHs from an infectious etiology to a traumatic etiology.⁶¹¹ And multiple studies have not only validated that premise⁶¹² but determined that trauma is actually the leading cause of SDHs in infants and children.⁶¹³ From years of research, physicians have compiled a list of additional potential causes of SDHs.⁶¹⁴ If one is to then study a particular subset (such as accidental trauma versus non-accidental trauma) of those causes, is not the only logical process to then attempt to exclude all other potential causes prior to studying that subset? This is the methodology that physicians have consistently employed in the various studies discussed above. And this is not “circular”; it is systematic and deductive.

Finally, Findley et al.’s critique of circularity itself suffers from a logical fallacy—the fallacy of hasty generalization. Findley et al. wish to place hundreds of studies into a box.⁶¹⁵ And they wish to paint, with one broad swath, a “CIRCULAR” sign along the side of that box, and set that box aside. *But are those studies, in fact, circular?* For example, Chadwick’s systematic review of short falls is not circular because a history of a short fall is not a criterion used to determine that a child has died.⁶¹⁶ Multiple studies of short falls avoid circularity by including EVERY fall seen at the respective institution in a given period of time and by not explicitly separating the children into groups of abused and non-abused children.⁶¹⁷ These

⁶¹¹ See Narang, *supra* note 10, at 523–29.

⁶¹² See *supra* Part 3; see also M. Shah et al., *Motor Vehicle Crash Brain Injury in Infants and Toddlers: A Suitable Model for Inflicted Head Injury?*, 29 CHILD ABUSE & NEGLECT 953, 954 (2005); Feldman et al., *supra* note 439, at 636; Hobbs et al., *supra* note 438, at 953; Tzoumi & Oates, *supra* note 438, at 1105; J. Urban et al., *Motor Vehicle Crash-Related Subdural Hematoma From Real-World Head Impact Data*, 29 J. NEUROTRAUMA 2774 (2012).

⁶¹³ See Feldman et al., *supra* note 439, at 638; Matschke et al., *supra* note 439, at 1587.

⁶¹⁴ See Narang, *supra* note 10, at 628.

⁶¹⁵ *Id.* at 596–627.

⁶¹⁶ See Chadwick, *supra* note 107.

⁶¹⁷ See Kravitz, Williams, Helfer, Lyons, Nimityongsukul, Levene, Ruddick, Schaefer, *supra* notes 51–62.

studies are manifestly non-circular because the measurement of the outcome variable does not even consider the predictor variable. Finnie's study made circularity impossible by randomly assigning specific lambs to be injured or not injured.⁶¹⁸ Multiple anthropometric and FEM studies use experimental designs that are simply not circular.⁶¹⁹ And the Odom study of the prevalence of RHs in the setting of chest compressions avoided circularity by considering only children thought not to be abused prior to their eye exam.⁶²⁰ These are but a few of the examples.

We are not interested in the elementary back-and-forth of: "yes, it is circular"—"no, it is not circular." The science speaks for itself. In hopes of transparency, and not "intimidation,"⁶²¹ we have presented a reasonable sample of that science. The scientific studies that form the basis of the scientific principles discussed above have been laid out for the reader to judge for himself/herself. For countless physicians, these studies and the principles they have produced are scientifically valid. They comprise the "major premise" of the AHT syllogistic argument.

2) What is the "differential diagnosis methodology?"

Stedman's Medical Dictionary defines "differential diagnosis" as "the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and contrasting of the clinical findings."⁶²² Simply stated, from a medical perspective, it is the list of diseases that physicians consider as possible causes for the signs or symptoms from which the patient is suffering.⁶²³ Interestingly, however, from a legal perspective, courts have varied in their interpretation and understanding of it. Some courts have interpreted it to be the methodology for arriving at causation by "ruling out" alternative causes.⁶²⁴ Others have interpreted it to require both a "ruling out" and "ruling in" process for

⁶¹⁸ See Finnie et al., *supra* note 242, at 237.

⁶¹⁹ See BIOMECHANICS, *supra* at 33–45.

⁶²⁰ See Narang, *supra* note 10, at 551, citing Amy Odom et al., *Prevalence of Retinal Hemorrhages in Pediatric Patients After In-Hospital Cardiopulmonary Resuscitation: A Prospective Study*, 99 PEDIATRICS, at *4 (June 1997).

⁶²¹ See Findley et al., *supra* note 3, at 296.

⁶²² Stedman's Medical Dictionary, 28th ed. (2006).

⁶²³ See Wong et al., *supra* note 406, at 691.

⁶²⁴ See *Bland v. Verizon Wireless, L.L.C.*, 538 F.3d 893, 897 (8th Cir. 2008) (stating that "a 'differential diagnosis [is] a technique that identifies the cause of a medical condition by eliminating the likely causes until the most probable cause is isolated.'"); *Wilson v. Taser Int'l, Inc.* 2008 WL 5215991, at *5 (11th Cir. Dec. 16, 2008) ("[N]onetheless, Dr. Meier did not perform a differential diagnosis or any tests on Wilson to rule out osteoporosis and these corresponding alternative mechanisms of injury. Although a medical expert need not rule out every

arriving at causation.⁶²⁵ And others have even created legal concepts, such as “differential etiology,” declaring it to be different from “differential diagnosis.”⁶²⁶

While courts have expressed variable levels of understanding of the differential diagnosis methodology, it is cognitive scientists who have provided the deepest and richest understanding of that methodology. Decades of research has revealed that, whereas it was once thought that physician clinical reasoning proceeded in a discretely linear fashion known as Bayesian analysis,⁶²⁷ the diagnostic process is actually a non-linear, unstructured method of problem-solving that employs both inferential and deductive reasoning.⁶²⁸ On occasion, and for various reasons (such as clinical exigency), physicians may bypass the hypothetico-deductive approach and utilize a different reasoning process known as “heuristics,” or problem-solving shortcuts.⁶²⁹

In the differential diagnosis methodology, the physician gathers historical information on a patient’s symptoms and signs and generates hypotheses (a.k.a., the differential diagnosis).⁶³⁰ Through the attainment of additional clinical information (via various diagnostic tests), the physician goes through an inferential and deductive process of hypothesis refinement until a consistent “working diagnosis” is achieved.⁶³¹ Hypothesis refinement utilizes a variety of reasoning strategies—probabilistic, causal, and deterministic—to discriminate among the

possible alternative in order to form an opinion on causation, expert opinion testimony is properly excluded as unreliable if the doctor ‘engaged in very few standard diagnostic techniques by which doctors normally rule out alternative causes.’”).

⁶²⁵ *Feit v. Great West Life & Annuity Ins. Co.*, 271 Fed. App’x. 246, 254 (3d Cir. 2008) (“Although this Court generally recognizes differential diagnosis as a reliable methodology, the differential diagnosis must be properly performed in order to be reliable. To properly perform a differential diagnosis, an expert must perform two steps: (1) ‘Rule in’ all possible causes of Dr. Feit’s death; and (2) ‘Rule out’ causes through a process of elimination whereby the last remaining potential cause is deemed the most likely cause of death.”).

⁶²⁶ See Wong et al., *supra* note 406, at 691.

⁶²⁷ “Bayesian analysis assembles a complete set of diagnostic hypotheses that can explain a given set of clinical findings. For each hypothesis, a set of relevant attributes is identified (historical findings, physical findings, complications, predisposing factors, laboratory results) that might help discriminate among the diagnoses. The prior probability of each diagnostic hypothesis is specified numerically, as is the probability that each attribute is found in each disease entity. Then, a calculation is made of the likelihood of each disease entity given the disease prevalence and the probability of each clinical attribute.” See Jerome P. Kassirer et al., *Learning Clinical Reasoning* (2d ed. 2009). Although physician reasoning does not exclusively proceed in a Bayesian fashion, physicians do frequently rely on Bayesian reasoning (combining disease prevalence with their knowledge of frequency of signs and symptoms in a given disease) in the diagnostic process; See also Wong et al., *supra* note 436 at 708.

⁶²⁸ See J. Kassirer and F. Sonnenberg, *The Scientific Basis of Diagnosis* in TEXTBOOK OF INTERNAL MEDICINE (WN Kelley, ed., 1989). See also Arthur S. Elstein and Alan Schwartz, *Clinical Problem Solving and Diagnostic Decision Making: Selective Review of the Cognitive Literature*, 324 BMJ 729, 730 (2002)

⁶²⁹ See Wong et al., *supra*, note 436 at 705-706.

⁶³⁰ *Id.* at 705.

⁶³¹ *Id.* at 706. The process of hypothesis refinement is an “evolving, sequential process of data gathering and interpretation.” See Kassirer et al., *supra* note 519 at 5-6.

existing diagnoses of the differential diagnosis.⁶³² While being mindful of the pitfalls of heuristics, the physician ultimately proceeds to hypothesis confirmation when the laws of diagnostic “adequacy,” “coherency,” and “parsimony” are satisfied.⁶³³ In the simplest sense, the methodology relies on process-of-elimination reasoning. As one eminent evidentiary scholar stated, “in differential diagnosis, if there are four possible diagnoses and you eliminate three, logic points to the last illness as the correct diagnosis.”⁶³⁴

In AHT/SBS cases, the differential diagnosis depends on the findings presented. Soft tissue injuries (such as bruises) have a differential diagnosis. Fractures (either long bone or skull) have a differential diagnosis. And intracranial findings (such as SDHs or cerebral edema) and ophthalmologic findings (such as RHs) also have a differential diagnosis.⁶³⁵ It is the physician’s task to parse through the historical information, the physical examination, and the laboratory and radiologic results to arrive at a unifying diagnosis that satisfies the criteria of “adequacy,” “parsimony,” and “coherency.” As Narang has already stated, in many cases but obviously not all, the unifying diagnosis will be trauma.⁶³⁶ From there, the physician will again utilize the historical information, the physical examination, the laboratory/radiology results, the medical literature, and his/her experience to distinguish between accidental and non-accidental trauma. This process/methodology will be examined in further detail below.

3) Is the “differential diagnosis methodology,” in general, a reliable methodology for applying the major premise to the specific facts of a case, i.e., the “minor premise”?

⁶³² See Kassirer et al., *supra* note 519 at 5-6. Probabilistic reasoning is Bayesian-type reasoning where prior probabilities of diseases are considered and combined with a physician’s knowledge of the frequency of signs and symptoms in a given disease and the probabilities of specific test information. These assist the physician in a probabilistic assessment of the most likely hypothesis. Causal reasoning is a “function of the anatomical, physiological and biochemical mechanisms that operate in normally in the human body and the pathophysiologic behavior of these mechanisms in disease.” In assessing causality, physicians use any reliable data, no matter the source. Additionally, temporal proximity can be a potent factor in assessing causation. See J Kassirer and J Cecil, *Inconsistency in Evidentiary Standards for Medical Testimony: Disorder in the Courts*, 288 JAMA 1382, 1384 (Sep 18, 2002),

⁶³³ “Adequacy” is when the remaining working hypothesis reasonably accounts for all the patient’s findings, both normal and abnormal. “Coherency” is when the patient’s findings are consistent with the altered pathophysiology of the hypothesized disease. “Parsimony” is the simplest explanation for all of the patient’s findings. See Wong et al., *supra* note 406 at 736; See also Kassirer et al., *supra* note 519, at 5-6.

⁶³⁴ Edward J. Imwinkelried, *The Admissibility and Legal Sufficiency of Testimony About Differential Diagnosis (Etiology): Of Under-and over-Estimations*, 56 BAYLOR L. REV. 391 (2004).

⁶³⁵ See generally Narang, *supra* note 10, at 628-629.

⁶³⁶ *Id.* at 573.

A scholar once mused that both Sherlock Holmes and Captain Spock agreed on the same proposition—“when you have eliminated the [other possibilities], whatever remains, however improbable, must be the truth.”⁶³⁷ That scholar went on to state, “when the most logical human and a Vulcan agree on a proposition, that proposition must have merit.”⁶³⁸ While this is not the crux of our analysis, it is an interesting starting point.

Courts have long held that the differential diagnosis methodology is a reliable methodology for arriving at specific causation in tort cases.⁶³⁹ In those cases, courts have also commented that “the differential diagnosis is a well-recognized and widely-used technique in the medical community to identify and isolate causes of disease and death.”⁶⁴⁰ In criminal cases, and more specifically in AHT/SBS cases, the courts have concluded no differently.⁶⁴¹ However, judicial decisions have offered little insight into *why* the methodology is reliable. Additionally, recent investigation into the forensic sciences has raised concern over implicit contextual and cognitive biases in the methodologies underlying forensic judgments.⁶⁴²

Thus, these concerns may justify a re-evaluation of previously unchallenged methodologies. With all due respect to the acumen of the brethren in the long black robe, the ipse dixit of the judicial expert may be no better than the ipse dixit of the medical expert. So then, what exactly is it that makes the differential diagnosis methodology reliable? And has it

⁶³⁷ See Imwinkelried, *supra* note 516 at 391.

⁶³⁸ *Id.*

⁶³⁹ See *Best v. Lowe’s Home Ctrs. Inc.*, 563 F.3d 171, 178, 183–84 (6th Cir. 2009) (stating a differential diagnosis can be adequate grounds for a causation opinion under *Daubert*); *Hyman & Armstrong, P.S.C. v. Gunderson*, 279 S.W.3d 93, 107 (Ky. 2008); *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 263 (4th Cir. 1999).

⁶⁴⁰ See *Gunderson*, 279 S.W.3d at 107 (citing *Globetti v. Sandoz Pharms. Corp.*, 111 F.Supp.2d 1174, at 1177 (N.D. Ala. 2000)).

⁶⁴¹ See *State v. McMullen*, 900 A.2d 103, at 118 (Del. Super. Ct. 2006) (holding that testimony of two state medical experts regarding Pediatric Condition Falsification was sufficiently relevant and reliable under *Daubert* when those experts “soundly performed” their differential diagnosis); *Overton v. State*, No. 13-07-00735-CR., 2009 WL 3489844 at *46 (Tex. App. Corpus Christi) (holding that trial court did not abuse its discretion in finding expert testimony opining that a child died of “non-accidental hypernatremia” reliable under *Daubert* when that expert based his opinion on “the widely accepted practice of differential diagnosis”); *State v. Edwards*, No. C–100200, 2011 WL 1378927 at *3 (Ohio Ct. App. 2011) (holding that the trial court did not abuse its discretion when it concluded expert testimony on AHT/SBS was reliable under *Daubert*; the court stated that “differential diagnosis is a standard scientific method for determining causation”); *State v. Carr*, No. C-090109, 2010 WL 2473337 at *6 (Ohio Ct. App. 2010) (stating that the “process of isolating the cause of a patient’s injuries through the methodical elimination of other potential causes, called differential diagnosis, is a standard scientific method for determining causation”, and expert testimony based upon such was reliable under *Daubert*).

⁶⁴² See National Research Council, *supra* note 409 at 4. Although clinical medicine is not considered a classic forensic discipline (such as fingerprint identification, forensic pathology, or bite-mark identification), there are aspects of clinical medicine, such as child abuse pediatrics, that have direct forensic applications. Thus, the concerns raised by the NRC are, at least, tangentially relevant to the methodologies employed by child abuse pediatricians.

been unsalvageably infected with the contextual and cognitive biases that seem to undermine other forensic judgments?

One oft-asserted validation of the differential diagnosis methodology is that it is a methodology employed for making life-or-death decisions, and ergo, must be reliable. The Advisory Committee Notes for the drafters of the Federal Rules of Evidence indicates that one of the drafters' objectives was to formulate evidentiary standards in accord with experts' practices in the field.⁶⁴³ Those Notes asserted that if a physician “in his own practice” considers a certain type of data and “makes life-and-death decisions in reliance on them,” common sense suggests that reasoning relying on such data should also be acceptable in the courtroom.⁶⁴⁴ Those same advisory committee comments were echoed almost a decade after the Federal Rules of Evidence went into effect by a leading evidentiary commentator, Professor Charles Nesson, when he published a celebrated article arguing that testimony based on the reasoning processes commonly used by medical diagnosticians ought to be admissible in court.⁶⁴⁵

Another factor pointing to its reliability is that the differential diagnosis methodology is rooted in the scientific method. If the core of science is “falsifiability”—the formulation of hypotheses and the conduct of systematic experimentation or observation to validate or “falsify” those hypotheses—then the differential diagnosis methodology is science embodied (no pun intended). Physicians formulate causal hypotheses for illness and other medical injuries and utilize the differential diagnosis methodology for the systematic validation or falsification of those hypotheses. But not only has the differential diagnosis employed “falsifiability,” it has been subjected to it as well. As mentioned above, cognitive scientists have conducted years of research on the methodology to assess and identify its strengths and weaknesses. In that course, the differential diagnosis methodology has itself been subject to peer-reviewed publication.⁶⁴⁶

Additionally, the methodology’s process-of-elimination reasoning is utilized not just by physicians, but by lay persons and lawyers as well.⁶⁴⁷ It is not just “generally accepted”; it is

⁶⁴³ See Imwinkelried, *supra* note 462 at 392.

⁶⁴⁴ *Id.* at 393.

⁶⁴⁵ *Id.*

⁶⁴⁶ See Kassirer, *supra* notes 479 & 480.

⁶⁴⁷ See Imwinkelried, *supra* note 462, at 420 (citing *Clausen v. M/V New Carissa*, 156 F. Supp. 2d 1192, 1194-95 (D. Or. 2001) (utilizing expert description of process-of-elimination reasoning to determine whether an oil spill caused the death of oysters in a commercial farm)).

“generally utilized.” As Karl Popper, one of the pre-eminent philosophers of science, himself stated, science is only “common-sense knowledge writ large.”⁶⁴⁸

The most reasonable challenge to the reliability of the differential diagnosis methodology (or to any methodology that involves human analytic reasoning) is *bias* and *errors of cognition*. Findley et al. have labeled some of these as “observer bias” and “interpretive errors” (such as “improper classifications” and “the prosecutor’s fallacy.”)⁶⁴⁹ The National Research Council, in its recent global assessment of the forensic sciences, generally described them as “cognitive” and “contextual” biases.⁶⁵⁰ The Council went on to warn that “the traps created by such biases can be very subtle, and typically one is not aware that his or her judgment is being affected.”⁶⁵¹ With regards to the forensic disciplines, the Council concluded that:

Unfortunately, at least to date, there is no good evidence to indicate that the forensic science community has made a sufficient effort to address the bias issue; thus, it is impossible for the committee to fully assess the magnitude of the problem.⁶⁵²

Thus, the Council recommended that the National Institute of Forensic Science fund and conduct further research on human observer bias and sources of human error in forensic examinations.⁶⁵³

In its discussion of error rates, the Council identified four statistical principles that are especially helpful in assessing error rates: sensitivity, specificity, positive predictive value, and negative predictive value.⁶⁵⁴ The Council stated that a “global error rate” can be estimated by summing the percentage of false positives and false negatives of a particular test.⁶⁵⁵ For example, a test with a 95% sensitivity has a 5% false negative rate.⁶⁵⁶ And a test with a 97% specificity has a 3% false positive rate.⁶⁵⁷ Thus, the “global error rate” of that particular test

⁶⁴⁸ *Id.* at 420 (citing Karl Popper, *The Logic of Scientific Discovery*, 22 (1959)).

⁶⁴⁹ See Findley et al., *supra* note 3 at 286-290.

⁶⁵⁰ See NATIONAL RESEARCH COUNCIL, *supra*, note 409, at 4-9.

⁶⁵¹ *Id.* at 6-2 (citing I.E. Dror and D. Charlton, *Why Experts Make Errors*, 56 J. FORENSIC IDENTIFICATION 600-616 (2006)).

⁶⁵² *Id.* at 8 (citing Giannelli, *Wrongful Convictions and Forensic Science: The Need to Regulate Crime Labs*, 86 N.C.L. REV. 163, 220-22 (2007)).

⁶⁵³ *Id.* at 6, 8.

⁶⁵⁴ *Id.* at 119, 20. Narang discussed these principles (along with odds ratios) in his first article. See Narang, *supra* note 10, at 538.

⁶⁵⁵ See NATIONAL RESEARCH COUNCIL, *supra* note 409, at 143.

⁶⁵⁶ The “sensitivity,” or the “true positive rate” (TPR) of a particular test, is the probability that a test for a disease will give a positive result when the patient actually has the disease. Put simply, it is actually the chance the condition will be found by the test. See Narang, *supra* note 10, at 538.

⁶⁵⁷ *Id.*

could be estimated as 4% $[(5+3)/200 \times 100=4 \text{ percent}]$. While a bit simplistic, and not dispositive of the reliability of a particular test, it does offer some statistical quantification of the “error rate” criteria sought by *Daubert*. Narang discussed the application of these statistical principles (and odds ratios) to the SDH/RH literature in his first article.⁶⁵⁸ The application of these statistical principles in the differential diagnosis methodology of a child abuse case will be exemplified below.

As mentioned above, cognitive scientists have provided invaluable insight on bias in decision-making, specifically in clinical medicine. In Daniel Kahneman’s Nobel prize-*acceptance* essay, *Maps of Bounded Rationality: A Perspective on Intuitive Judgment and Choice*, the author summarizes a long and arduous journey wherein he and his colleague, Amos Tversky, “explored the psychology of intuitive beliefs and choices and examined their bounded rationality.”⁶⁵⁹ Kahneman and Tversky identified “a two-system view” that distinguishes intuition from reasoning..⁶⁶⁰ “The operations of System 1 [intuition] are fast, automatic, effortless, associative, and difficult to control or modify.”⁶⁶¹ “The operations of System 2 [reasoning] are slower, serial, effortful, and deliberately controlled; they are also relatively flexible and potentially rule-governed.”⁶⁶² “A defining property of intuitive thoughts is that they come to mind spontaneously, like percepts,” and can result in rash judgments, also known as “heuristics.”⁶⁶³ This concept of “heuristics” has been a vital concept in understanding the pitfalls of clinical decision-making.⁶⁶⁴

The concept of “judgment heuristics” informs us that “intuitive judgments of probability are mediated by attributes such as similarity and associative fluency” and “are not intrinsically related to uncertainty.”⁶⁶⁵ Other attributes of heuristic judgments include susceptibility to availability bias, accessibility bias, anchoring, representativeness, overweighting, and attribute

⁶⁵⁸ *Id.* at 542-56.

⁶⁵⁹ Daniel Kahneman, Lecture at the Princeton University Dep’t of Psychology: Maps of Bounded Rationality: A Perspective on Intuitive Judgment and Choice, 449 (Dec. 8, 2002), *available at* http://nobelprize.org/nobel_prizes/economics/laureates/2002/kahneman-lecture.pdf.

⁶⁶⁰ *Id.* at 450-51.

⁶⁶¹ *Id.* at 450.

⁶⁶² *Id.*

⁶⁶³ *Id.* at 452, 455.

⁶⁶⁴ See Kassirer, *supra* notes 479 & 480, at .

⁶⁶⁵ See Kahneman, *supra* note 511, at 455.

substitution, to name a few.⁶⁶⁶ Kahneman notes that “people rely on a limited number of heuristic principles which reduce the complex tasks of assessing probabilities and predicting values to simpler judgmental operations.”⁶⁶⁷ While these heuristics can be quite useful, “. . . sometimes they lead to severe and systematic errors.”⁶⁶⁸

However, Kahneman and Tversky determined that errors of heuristic judgments can be tempered by the slow, serial, effortful, deliberate, and rule-oriented operations of System 2.⁶⁶⁹ The efficacy of System 2, in its ability to mitigate heuristic judgments, is impaired “by time pressure,” “by concurrent involvement in a different cognitive task,” and even “by being in a good mood.”⁶⁷⁰ The authors state:

The central finding in studies of intuitive decisions, as described by Klein (1998), is that experienced decision makers working under pressure, such as captains of firefighting companies, rarely need to choose between options because in most cases only a single option comes to their mind. . . . *Doubt is a phenomenon of System 2, a meta-cognitive appreciation of one’s ability to think incompatible thoughts about the same thing.*⁶⁷¹

“Conversely, the facility of System 2 is positively correlated with intelligence, with ‘need for cognition,’ and with exposure to statistical thinking.”⁶⁷² Thus, the question to be asked is: *how susceptible is the differential diagnosis methodology in child abuse cases to heuristic judgments?* It would be scientifically irresponsible to state that the differential diagnosis methodology employed in child abuse cases is immune to heuristic judgments. However, several factors make it less susceptible. First and foremost, the exigency of “time-pressure” circumstances is not one encountered by the child abuse pediatric consultant. Unlike the firefighter, the emergency room physician treating a patient suspected of a myocardial infarction (a.k.a., heart attack) or the intensive care physician running “a code, the child abuse pediatric consultant does not experience those time-pressured circumstances. Like other diagnostic consultants, such as infectious disease or endocrinology, the child abuse pediatric consultant has

⁶⁶⁶ *Id.* at 465; see also Arthur Elstein & Alan Schwartz, *Clinical Problem Solving and Diagnostic Decision Making: Selective Review of the Cognitive Literature*, 324 BRIT. MED. J. 729 (2002).

⁶⁶⁷ Kahneman, *supra* note 522, at 465.

⁶⁶⁸ *Id.* (citing Amos Tversky & Daniel Kahneman, *Judgment under Uncertainty: Heuristics and Biases*, 185 SCI. 1124–1131 (1974)).

⁶⁶⁹ Kahneman, *supra* note. 522, at 450.

⁶⁷⁰ *Id.* at 473.

⁶⁷¹ *Id.* at 455–56 (emphasis added).

⁶⁷² *Id.* at 473 (citations omitted).

hours to days (if not weeks, in certain circumstances) to cogitate upon a differential diagnosis, to order appropriate laboratory and radiology tests, to confer with other subspecialists and interdisciplinary partners, and consequently, to further refine that differential.

Second, the lack of time pressure naturally creates an environment that is suited to a “need for cognition.” The child abuse pediatric consultant has ample opportunity and resources for the creation and resolution of doubt. But as Kahneman and Tversky warn, bias is often implicit and goes unrecognized.⁶⁷³ So what assurance is there that even the cogitating, unpressured child abuse consultant is not shackled with implicit biases?

There are two other important attributes that mitigate the impact of any implicit biases—the multi-disciplinary approach and the utilization of statistical thinking/EBM (evidence-based medicine). Child abuse pediatric consultants often engage in multi-disciplinary evaluations of child abuse cases. These evaluations involve the cognitive efforts of multiple pediatric subspecialists—radiologists, ophthalmologists, neurosurgeons, hematologists, orthopedic surgeons, pathologists, and child abuse pediatricians. Additionally, social interdisciplinary partners (such as law enforcement officials and social workers) provide valuable information that typically is not obtained in routine medical history gathering. Thus, a system is forged whereby comprehensive information is collectively gathered, shared, and evaluated. And as mentioned above, this is not a process amenable or available to all clinical situations and all circumstances (i.e., the ER physician in an emergency situation or the critical care physician in a critical situation). Is this an error proof process? Of course not. Is this an indictment of particular physicians? Of course not. It is merely a recognition of the fact that the comprehensive and collective cognitive operations of the many at least minimize the risk of undetected implicit bias in the single.

Finally, as demonstrated in Narang’s first article and in the sections above, physicians have utilized EBM to improve the scientific data in child abuse research. As one cognitive scientist stated: “Evidence based medicine is the most recent, and by most standards the most successful, effort to date to apply statistical decision theory in clinical medicine.”⁶⁷⁴

Physicians have incorporated rigorous design methodologies and statistical analyses (such as logistic regression) to account for confounding variables and bias. These have resulted

⁶⁷³ *Id.* at 465.

⁶⁷⁴ See Elstein & Schwartz, *supra* note 518, at 731.

in not only scientific principles with high degrees of statistical confidence and probability but in principles that have been reproduced, along multiple lines of research, by various physician scientists across the world. As Narang discussed in his first article, the concept of convergent validation offers explanation for their increased validity.⁶⁷⁵ Kahneman echoes this in his conclusion: “The claim that cognitive illusions will occur unless they are prevented by System 2 sounds circular, but it is not. Circular inferences are avoidable because the role of System 2 can be independently verified in several ways.”⁶⁷⁶

Despite these insulations, is there room for improvement in minimizing the potentiality of implicit bias in clinical decision-making? Certainly. Probably the most important one is the need for increased recognition of and education about the topic.⁶⁷⁷ As judges have recently increased awareness and education on implicit bias in judicial decision-making, physicians need to follow suit. Currently, early medical education involves some “problem-based learning” on the formulation and testing of clinical hypotheses.⁶⁷⁸ But detailed, intensive education about the pitfalls of clinical decision-making—i.e., the pitfalls in “hypothesis generation,” “diagnostic creation and revision,” and “probability estimation and revision”—is lacking.⁶⁷⁹ Thus, increased education in medical school and continuing medical education would be good steps in this direction.

Another consideration in assessing the reliability of the differential diagnosis methodology in AHT/SBS cases is whether it is tethered to the standards of medical practice. Is the same methodology employed in AHT/SBS cases as in other medical cases? Or is it a peculiar diagnostic methodology? In Narang’s first article, and herein above, Narang argued that the methodology employed in coming to the diagnosis of AHT is no different than the methodology employed in arriving at *any* medical diagnosis.⁶⁸⁰ Findley et al. have asserted that the medical decision-making in AHT/SBS cases differs from other medical diagnoses, such as migraine headaches, because in those diagnoses, unlike in AHT/SBS, “doctors generally correlate the

⁶⁷⁵ See Narang, *supra* note 10, at 579.

⁶⁷⁶ Kahneman, *supra* note 522, at 482.

⁶⁷⁷ See Elstein & Schwartz, *supra* note 518, at 731-32.

⁶⁷⁸ *Id.* at 731.

⁶⁷⁹ *Id.* at 729-31.

⁶⁸⁰ Narang, *supra* note 10, at 571-74.

patient's description of the symptoms and their onset (the patient history) with objective medical data (such as lab results) and response to treatment.”⁶⁸¹

This assertion is, in a word, wrong. As has been demonstrated above, physicians *do* order a host of laboratory and radiologic tests (bleeding studies, bone health labs, x-rays, CTs, MRIs, etc.) and correlate those results with a patient's history. Physicians *do* effect a treatment—placement in a safer environment—and assess the reoccurrence of any symptoms or injuries in that safer environment. In fact, it is the very inconsistency of the history provided by a caregiver with these objective results that is the cornerstone of the AHT/SBS diagnosis. So the more pertinent question is whether there are other medical diagnoses where there is inconsistency between history and objective medical data and the differential diagnosis methodology is also employed in arriving at that diagnosis.

The answer is, in a word, yes. There are multiple diagnoses that fit this bill—pediatric condition falsification, anorexia nervosa, and drug seeking behavior, to name a few. However, a detailed analysis of one should crystalize this point. The diagnosis of bulimia nervosa is “binge eating and inappropriate compensatory methods to prevent weight gain.”⁶⁸² “The most common compensatory technique is the induction of vomiting after an episode of binge eating,” but “[o]ther purging behaviors include the misuse of laxatives and diuretics.”⁶⁸³ A key component of the diagnosis is the patient's denial of the purging behavior, but with manifest physical signs or lab tests indicating the diagnosis. Some of these findings include dental erosion, palatal or oral trauma (from attempted induction of vomiting), abrasions along the backs of the hands (from attempted induction of vomiting), or electrolyte imbalances (from chronic vomiting or laxative use).⁶⁸⁴ As with any other medical condition, there are other conditions on the differential diagnosis to consider prior to arriving at the diagnosis. These include anorexia nervosa, depression, gastrointestinal obstructive disorders, body dysmorphic disorder, Kluver Bucy syndrome, and gastrointestinal infectious diseases.⁶⁸⁵ It is the physician's task to consider these other disorders on the differential, and order the appropriate labs and imaging prior to ruling

⁶⁸¹ Findley et al., *supra* note 3, at 281.

⁶⁸² AM. PSYCHIATRIC ASS'N, DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS 589 (4th ed., text revision 2000).

⁶⁸³ *Id.* at 590.

⁶⁸⁴ *Id.* at 592.

⁶⁸⁵ *Id.* at 593-94.

them out⁶⁸⁶ and arriving at the bulimia nervosa diagnosis. There is no question that bulimia nervosa is a valid diagnosis, or that a physician can reliably arrive at that diagnosis using the differential diagnosis methodology.

Much of what has been discussed above has been conceptual, esoteric, and possibly even vague. Perhaps an example of how the physician employs the differential diagnosis methodology in an AHT/SBS case will assist in clarifying the issue of its reliability. A 3 month-old infant presents to the emergency room for “stopping breathing” (apnea). The mother’s boyfriend, who was caring for the child while the mother was at work, states that the infant was crying. When he gave the infant a bottle, the infant “choked and gagged” and then “stopped breathing.” He “shook” the infant gently to revive the infant. When the infant began crying a short time later, he soothed the infant and waited for the mother to return home, which occurred some hours later. When the mother returned home some hours later, the infant appeared pale and lethargic, and so the mother and her boyfriend proceeded to the ER for evaluation.

At the ER, the mother and her boyfriend denied *any* trauma for the infant in the prior 3 months of life. The mother denied any other problems in the child’s medical history or any notable family medical history. On physical examination, the child was noted to have a small amount of swelling to the back of the head, but nothing else notable on physical examination—no bruising, scars, or other lesions. A head CT scan performed in the ER revealed an acute (fresh) subdural hemorrhage (SDH) along the front of both brain hemispheres and in between them (interhemispheric) and developing cerebral edema (brain swelling). The child was admitted for further evaluation and management, and CPS was called. Further hospital evaluation, including whole body x-rays (a skeletal survey), revealed healing rib fractures on the right side of the rib cage. Ophthalmologic exam by the pediatric ophthalmologist revealed severe retinal hemorrhages (RHs) in both eyes. A child abuse pediatrician was consulted.

In this scenario, which is not different from many child abuse cases, the child abuse pediatrician is presented with multiple findings⁶⁸⁷—soft tissue swelling to the head, acute SDH,

⁶⁸⁶ “Individuals whose binge-eating behavior occurs only during Anorexia Nervosa are given the diagnosis Anorexia Nervosa, Binge-Eating/Purging Type, and should *not* be given the additional diagnosis of Bulimia Nervosa.” *Id.* at 593 (emphasis in original). However, in certain neurological or other general medical conditions, if the full criteria for Bulimia Nervosa is also met, both diagnoses can be given. *Id.*

⁶⁸⁷ Many critics lump AHT/SBS cases into “triad” cases (SDH, RH, and cerebral edema). However, this is oversimplistic and inaccurate. While certainly a small percentage of AHT/SBS cases contain only these findings, in many other cases there are other important findings which physicians must account for in the unifying diagnosis.

brain swelling (cerebral edema), severe RHs, and rib fractures—all of which have their own differential diagnoses.⁶⁸⁸ For example, soft tissue swelling of the scalp has a limited differential diagnosis—trauma, infection (such as fungal or bacterial), inflammatory conditions, and dermatologic conditions (such as epidermal inclusion cysts). SDHs and RHs have a more expansive differential,⁶⁸⁹ which can be generally characterized as trauma, bleeding disorders, malignancy, infection, and metabolic/genetic diseases. Rib fractures have a limited differential—trauma, genetic disease (such as osteogenesis imperfecta), nutritional deficiency (which results in weakened bone health and predisposes bones to fracture with mild trauma), prematurity, and medical procedures (such as cardiopulmonary resuscitation).⁶⁹⁰ And cerebral edema does not itself have a differential diagnosis, but is rather a complex pathophysiological response to brain injury (either resulting primarily from direct trauma or as a secondary response to lack of blood oxygen and blood).⁶⁹¹

*After the formulation of appropriate differential diagnoses for the relevant medical findings, it is at this point that the child abuse pediatrician engages in an inferential and deductive reasoning process that is in some aspects Bayesian, and some aspects not.*⁶⁹² In ruling out certain conditions on the differential, a physician may only utilize historical information, such as whether or not the child was born prematurely to rule out “prematurity,” or whether or not CPR was performed to rule out “medical procedures” (as differential diagnoses for the rib fractures). In other cases, the physician may utilize historical information in combination with physical exam findings to rule out certain conditions. For example, in ruling out fungal or bacterial infection (as differential diagnoses for the soft-tissue swelling on the head), a physician would utilize the presence or absence of fever and the presence or absence of physical exam skin findings indicative of infection (like redness, warmth, boggiess, or the presence of blisters, vesicles, or scales). And, in other cases, the physician would combine historical information, physical exam findings, and laboratory information to rule out other conditions on the

⁶⁸⁸ It is important to note at this point that, for the purposes of simplicity in this example, the determination of the medical findings is not disputed, as can be in real-life circumstances. For example, whether a radiographic finding represents a true fracture or is a normal variant of the human body or some other explanation is another consideration in medical decision-making that must be made and involves training, experience, and ongoing literature review.

⁶⁸⁹ Narang, *supra* note 10, at 628-29.

⁶⁹⁰ See Reece, *supra* note 17, at 148-49.

⁶⁹¹ *Id.* at 103.

⁶⁹² See Kassirer, *supra* notes 479-80.

differential—such as using the absence of any historical symptoms, physical findings of lymph nodes or liver or spleen enlargement, and a normal white blood cell count (a lab) to rule out leukemia (on the differential for SDHs and RHs).

All these manners of eliminating certain conditions from the various differential diagnoses are non-Bayesian, i.e., there is no statistical quantification (pre and post-test probabilities) of the singular (e.g., the presence or absence of premature history in eliminating “prematurity”) or cumulative (e.g., using historical, physical exam information, and the white blood count to eliminate “leukemia”) probabilities of these diagnostic factors in the diagnostic process. Yet these are *very reliable* methods of eliminating some of the conditions from the differential diagnosis. They are rooted in years of experience with the known pathophysiological processes of disease and the human body. And often, they are sanctified in medical treatises.⁶⁹³ This is simply a reminder that the mere absence of linear, Bayesian analysis does not connote unreliability.

But there are aspects of the AHT/SBS differential diagnosis methodology that are conducive to Bayesian analysis. For example, in order to rule out bleeding disorders as a possible cause of SDHs in our scenario, it is informative to calculate the probability that a given bleeding disorder causes intracranial hemorrhage (ICH) in the general population. If we remember from the Bleeding Disorders section above, this is attainable by multiplying the prevalence of that bleeding disorder by the prevalence of ICH in that bleeding disorder:⁶⁹⁴

(Prevalence of bleeding disorder) x (Prevalence of ICH in that bleeding disorder).
For example, as noted in the Bleeding Disorders section above, the prevalence of Hemophilia A (Factor VIII) in the male population is 1 in 5000.⁶⁹⁵ Also as noted in that section, the literature demonstrates that between 5–12% of these individuals will get an ICH at some time in their life.⁶⁹⁶ *Thus, assuming the highest prevalence (12%), the estimated probability that a person will get an ICH due to Hemophilia A is 2.4 per 100,000.* But that is a *lifetime* risk assessment for hemophiliacs. If even 10% of these intracranial hemorrhages occurred in the first year of life, the rate would be only 24.1 in every 100,000 boys (or about 1 in 4 million). *This is substantially lower than the incidence of SDHs attributable to AHT/SBS in the first year of life—which ranges*

⁶⁹³ See R. Behrman, NELSON’S TEXTBOOK OF PEDIATRICS (17th ed. 2004).

⁶⁹⁴ See *supra* Part 4.D.

⁶⁹⁵ See *supra* Table 1.

⁶⁹⁶ *Id.*

from 24-29/100,000.⁶⁹⁷ Comparatively, SDHs are about 100 times more likely to be secondary to AHT/SBS than Hemophilia A. And this is assuming the child has Hemophilia A. If lab testing rules out this diagnosis, then this comparative analysis is moot. Similar comparative ratios are available for all the bleeding disorders based upon the table listed in the Bleeding Disorders section above.⁶⁹⁸

Another example of Bayesian analysis is the probability of differentiating accidental trauma from AHT based upon the presence of the severe RHs. Here again, the evidence-based literature is instructive for physicians. Maguire et al. conducted a systematic review of RHs in AHT and accidental trauma.⁶⁹⁹ The authors used strict inclusion criteria to identify 62 studies that represented 998 children aged 0 to 11 years, many of which were comparative studies between cohorts of accidental and non-accidental injury patients.⁷⁰⁰ The authors only included studies where abuse was either witnessed, admitted, or confirmed through multidisciplinary assessment and, in the comparative studies with accidents, where the accidents were witnessed.⁷⁰¹ This ensured minimization of ‘circularity’ in diagnosis by not relying on clinical features in the diagnostic assessment.⁷⁰² The authors performed multilevel logistic regression so that the data would be “more strongly correlated within the studies than between the studies.”⁷⁰³ Based upon their meta-analysis, they found that the probability of abuse in a child with head trauma and RHs was 91%.⁷⁰⁴

However, rather than computing statistics (the probability of abuse) that were limited to the population of patients in their study, the authors also calculated an odds ratio of abuse—a statistic that is generalizable to all populations of patients.⁷⁰⁵ *The authors determined that a child with head trauma and RHs has an odds ratio of 14.66 (95% CI: 6.39-33.62) for abuse.*⁷⁰⁶

⁶⁹⁷ Christopher J. Hobbs et al., *Subdural Haematoma and Effusion in Infancy: An Epidemiological Study*, 90 ARCH. DIS. CHILD 952, 952 (2005); Heather T. Keenan et al., *A Population-Based Study of Inflicted Traumatic Brain Injury in Young Children*, 290 J. AM. MED. ASS’N 621, 621 (2003); Katherine D. Ellingson et al., *Using Hospital Discharge Data to Track Inflicted Traumatic Brain Injury*, 34 AM. J. PREV. MED. S157, S157 (2008).

⁶⁹⁸ See *supra* p. XX.

⁶⁹⁹ Sabine A. Maguire et al., *Retinal Haemorrhages and Related Findings in Abusive and Non-Abusive Head Trauma: A Systematic Review*, 27 EYE 28 (2013).

⁷⁰⁰ *Id.* at 28-30.

⁷⁰¹ *Id.* at 29.

⁷⁰² *Id.*

⁷⁰³ *Id.* at 29-30.

⁷⁰⁴ *Id.* at 31.

⁷⁰⁵ *Id.*

⁷⁰⁶ *Id.*

In other words, if you know what the likelihood of abuse is for children admitted to your own Pediatric Intensive Care Unit (PICU), ER, or clinic with trauma, and you then find that one of those children has RHs, the likelihood that the child has been abused is now 14.6 times greater than that prior probability.

And, these are not the only evidence-based statistics that physicians consider. Well-conducted systematic reviews discriminating inflicted from accidental injury have concluded that in a child with intracranial injury, *apnea has an odds ratio of 17 for abuse* (with a positive predictive value of 93%) and *rib fractures have an odds ratio of 3 for abuse*.⁷⁰⁷ While it may seem that these ratios (odds ratio of 14, 17, or 3) are numerically not that high, they are actually rather impressive. For example, there is a proven benefit that psychosocial interventions (such as self-help material and telephone support) help people with ischemic heart disease quit smoking.⁷⁰⁸ The evidence was so compelling for these strategies that they are now routine practice. Yet, the odds ratio for behavioral therapies was only 1.69 (95% CI 1.33 to 2.14), for telephone support 1.58 (95% CI 1.28 to 1.97), and for self-help only 1.48 (95% CI 1.11 to 1.96).⁷⁰⁹ Taken in this context, the odds ratio for abuse, with specific associated clinical features (apnea, retinal hemorrhages), are extremely compelling.

Finally, and most importantly, evidence-based literature has quantitatively specified that which common sense has qualitatively known: *in a child with traumatic intracranial injury, the absence of a trauma history has a 97% specificity and 92% positive predictive value for abuse*.⁷¹⁰ A predictive value is another statistic which is immensely helpful. It is the probability of a disease after additional information (such as from a test) has been obtained.⁷¹¹ Thus, a positive predictive value is the probability of disease in those known to have a positive test result. Or in the above scenario, in the child with traumatic intracranial injury, the absence of trauma history (a.k.a., the positive test) indicates a 92% probability of abuse.

While it may seem that the statistics are dispositive, they are actually only a portion of the analysis. Physicians must remain mindful of “conjunction fallacies,” “overweighting,”

⁷⁰⁷ Sabine A. Maguire et al., *Which Clinical Features Distinguish Inflicted from Non-inflicted Brain Injury? A Systematic Review*, 94 ARCH. DIS. CHILD 860, 860 (2009).

⁷⁰⁸ J. Barth et al., *Psychosocial Interventions for Smoking Cessation in Patients with Coronary Heart Disease*, COCHRANE DATABASE OF SYSTEMATIC REVIEWS 2008, Issue 1. Art. No.: CD006886.

⁷⁰⁹ *Id.*

⁷¹⁰ *Id.*

⁷¹¹ *See*

“errors in [the] revision of probabilities,” and other pitfalls.⁷¹² Cognizance of these potential errors minimizes the probability of their occurrence.

Ultimately, the differential diagnosis methodology is a marriage of evidence-based literature and experience; a symbiosis of inferential and deductive reasoning; a synergy of linear and non-linear dynamic thought. It is the methodology by which physicians achieve diagnostic sufficiency.⁷¹³ So with the information presented, the question for the reader and the gate-keeper is: Is the methodology presented “junk science”? Is it not even “good enough to be wrong”?

The reliability of the differential diagnosis methodology, a methodology utilized by all physicians—not just child abuse pediatricians, has been laid out for the reader to judge for himself/herself. For countless physicians, this methodology is reliable. In general, the application of this methodology to the facts of a case comprises the “minor premise” of the AHT syllogistic argument. The sound application of this methodology to a particular set of facts is for the individual, particular gate-keeper to determine.

C. The “Path Forward”: Throwing the baby out with the bath water (figuratively speaking)

Findley et al. have suggested that our disagreement on this issue (AHT/SBS) is “narrow but critical.”⁷¹⁴ We could not disagree more. Our disagreement lies not just in a misunderstanding of the quality or sufficiency of the medical literature; it represents a vast philosophical and ideological difference about the value and roles of clinical judgment and our current jury system in cases involving medical expert testimony. We shall examine the logical shortcomings of Findley et al.’s positions and discuss our recommendations for resolving *Daubert* issues in AHT/SBS cases.

1) The fog of legal argument

⁷¹² See Elstein and Schwartz, *supra* note 518, at 731-32.

⁷¹³ The standard for achieving diagnostic sufficiency is undefined. Whether it is a preponderance standard, clear and convincing standard, or beyond a reasonable doubt standard has never been carefully explored or clearly enunciated, in the medical or legal literature. In most circumstances, given clinical exigency and the primacy of treating the patient, the standard most likely approximates a preponderance standard. But this is a topic for further discussion at a different time. What is clear is that Findley et al., and others, have confused it for the standard for legal sufficiency for conviction—beyond a reasonable doubt.

⁷¹⁴ See Findley et al., *supra* note 3, at 215.

There is an old defense adage: “If you can’t win on the facts, argue the law; if you can’t win on the law, then just confuse everyone.” The applicability of this axiom will hopefully be apparent.

Findley et al. bemoan the “subjective” and unreliable nature of clinical judgment. The authors state that its “subjective nature” is not “the objective medical evidence envisioned by evidence-based medicine and *Daubert*” and would ultimately “result in mistaken diagnoses and false convictions.”⁷¹⁵ Despite making this assertion *repeatedly*,⁷¹⁶ the authors then conclude that certain clinical judgments (in AHT/SBS cases) are “obvious,”⁷¹⁷ without defining by what criteria they become “obvious,” if clinical judgment is “obviously” unreliable. Additionally, if “subjectivity” spells the death of clinical judgment, then what physician testimony could ever survive? Such reasoning would necessitate the undesirable exclusion of *all* physician testimony—the pediatrician, the emergency room physician, the neurosurgeon, the ophthalmologist, the radiologist, and the forensic pathologist.

Findley et al. invoke this same fallacy—hasty generalization—in their argument for the invalidation of the AHT/SBS diagnosis. The authors spend considerable effort arguing that biomechanics has conclusively demonstrated that shaking does not even come close to reaching the thresholds for causing concussive injury or SDHs.⁷¹⁸ Then Findley et al. generalize this premise—that shaking cannot cause SDHs—as the basis for invalidating the *entire* AHT/SBS diagnosis.⁷¹⁹ Yet, despite proffering the diagnosis as invalid, as mentioned above, the authors nevertheless concede that some AHT/SBS cases are “obvious.”⁷²⁰ This begs the question of how some cases are “obvious” if the diagnosis is in fact invalid.

Interestingly, the confusion takes a different turn. Whereas Findley et al. are adamant that science has demonstrated that shaking is not dangerous, the authors then ultimately concede that “violent shaking” is “dangerous,” and that “there are few costs and many potential benefits associated with educating parents that they should never shake a child.”⁷²¹ With that inconsistency being apparent, the question arises: If shaking does not even come close to

⁷¹⁵ *Id.* at 292.

⁷¹⁶ *Id.* at 216, 266, 292, 300.

⁷¹⁷ *Id.* at 301.

⁷¹⁸ *Id.* at 236-37.

⁷¹⁹ *Id.*

⁷²⁰ *Id.* at 301.

⁷²¹ *Id.* at 301.

concussive or SDH-causative forces, then what exactly do Findley et al. believe shaking is “dangerous” enough to cause? A bruise? A neck sprain? If shaking is not dangerous enough to cause serious injury, then why are prevention programs necessary or recommended? To prevent a potential bruise or neck sprain? Considerable medical research resources have already been spent to validate something that *res ipsa loquitur* sufficiently explains—that shaking a young infant can and does cause serious intracranial injury. It is careful, deliberate, and reasoned medical judgment through the differential diagnosis methodology which helps to eliminate potential alternative causes for those injuries.

2) Lifting the fog

Married with this confusion is a healthy distrust of the jury system. Findley et al. state: This approach [experts with differing perspectives arguing it out in the courtroom] presents two problems. First, trying and retrying undecided scientific issues on a weekly basis is extraordinarily expensive and inevitably results in inconsistent and ‘fluky’ justice. Second, and perhaps more important, if doctors cannot agree on these complex and unresolved issues, it is unlikely that jurors or judges can do any better.⁷²²

Findley et al. argue that the resolution to this issue, or “the path forward” or “getting it right,” is to “acknowledge the complexities,” and, when applicable, to say “we don’t know.”⁷²³ Setting aside the veiled professional ad-hominem that physicians do not already do this (and consequently, are diagnosing and testifying carelessly, capriciously, maliciously, or at best, erroneously), the logical consequence of this course is diagnostic and testimonial abstinence. But is that really “getting it right?” Or is that just a safer course for those who are alleged or actual perpetrators of abuse? Does diagnostic and testimonial abstinence not then end up supplanting one “fluky justice” for another? It seems to us that Justice should balance the “actual innocents” on both sides of the scale, not just those potentially accused of child abuse.

Thus, before we “toss the baby out with the bathwater,” we propose improvements in our current constructs, both medical and legal, as a true path forward. From the medical construct,

⁷²² *Id.* at 305.

⁷²³ *Id.* at 309-312.

first and foremost, there *must be* increased funding for child maltreatment research at both the state and federal levels.⁷²⁴ Increased funding for evidence-based research in the forensic disciplines was a primary recommendation by the National Research Council.⁷²⁵ The extension to the field of child maltreatment is a natural corollary. At present, the Institute of Medicine is already investigating avenues of needed research in the field of Child Maltreatment.⁷²⁶ However, it cannot be emphasized enough that the need for further research is not an indictment of the quality of the current research. As demonstrated above, multiple research collaboratives have enhanced the current evidence-based literature such that we are on the verge of clinical decision rules in AHT/SBS.⁷²⁷

Second, as Narang mentioned in his first article⁷²⁸ (and Findley et al. have echoed),⁷²⁹ a multidisciplinary body, under the auspices of the National Academy of Sciences and/or the National Institute of Health, needs to make a global assessment of the evidence-based literature on AHT/SBS and promulgate its findings. Third, medical professional societies must take a more active role in the regulation of irresponsible testimony by its members. One organization, the American Association of Neurologic Surgeons, has been a model for the societies in imposing disciplinary actions against its members for irresponsible expert testimony.⁷³⁰ Other societies need to follow suit. Finally, physicians need to engage in reciprocal intra-disciplinary and inter-disciplinary educational efforts on multiple medico-legal topics in AHT/SBS, to include, but not limited to, implicit bias in medical decision-making, evidence-based literature and AHT/SBS, and responsible expert testimony in AHT/SBS.

With regards to the legal construct, making inroads into the problem of ensuring reliable medical expert testimony requires travel along several paths. The Supreme Court and legal scholars have clearly recognized that *Daubert* has not turned out to be the panacea it was believed it would be. Thus, with the recent ruling in *Melendez-Diaz*,⁷³¹ some scholars assert the

⁷²⁴ While Findley et al. also recommend “research” as a path forward (see Findley et al., *supra* note 3, at 307-08), we agree with some of their delineated research objectives, but disagree with others (such as the establishment of a national registry of AHT/SBS cases).

⁷²⁵ See National Research Council report, *supra* note 409.

⁷²⁶ Email communications on file with Dr. Narang.

⁷²⁷ See Hymel et al., *supra* note 126.

⁷²⁸ See Narang, *supra* note 10, at 594.

⁷²⁹ See Findley et al., *supra* note 3, at 309.

⁷³⁰ See *Austin v. Am. Ass’n of Neurological Surgeons*, 253 F.3d 967 (7th Cir. 2001).

⁷³¹ See *Melendez-Diaz v. Mass.*, 557 U.S. 305 (2009).

Court has sought to remedy *Daubert*'s shortcomings by strengthening the confrontation rights of the accused.⁷³² While emboldening the Confrontation Clause is certainly an acceptable adjunct, emboldening the gatekeeper and the jury are valuable objectives as well. One important avenue for achieving those objectives is the increased utilization of FRE 706. The assistance of the independent expert to the court (for *Daubert* purposes) and to the jury (for any questions that may arise in the course of the adversarial process) could well serve the goals of truth and justice. This sage counsel was offered by Judge Learned Hand over a hundred years ago,⁷³³ but this counsel has found spotty adherence at best.

As with the medical construct, another important consideration is the need for more legal/social science research. Bold assertions have been made of a judicial system that is "riddled with false convictions."⁷³⁴ But in order to adequately assess the validity of such statements, or at least to adequately grasp the scope of the problem, a denominator is needed. Certainly, false convictions have occurred. But in what percentage of cases? Currently, there is only conjecture. Not only is that research needed, but further research is needed into why these false convictions have occurred, what variables are most common in those cases, and what systemic changes would be effective in minimizing those cases. Finally, also as with medicine, additional education for the gatekeeper (on topics such as the basics of good scientific studies, ongoing advances in the science related to AHT/SBS, etc.) would be beneficial.

Conclusion

What has been presented for the reader, in Narang's first article and in this article, is a reasonable summary of the evidence-based literature that girds the AHT/SBS diagnosis, an analysis of the strengths and limitations of that literature, and a detailed *Daubert* analysis of the differential diagnosis methodology by which physicians arrive at that diagnosis. These comprise the major and minor premises of the syllogistic structure of the expert's opinion offered in court. For countless physicians and professional medical societies, they are valid and reliable. What

⁷³² See Joelle Moreno, *C.S.I. BULLS#IT: The National Academy of Sciences, Melendez-Diaz v. Massachusetts, and Future Challenges to Forensic Science and Forensic Experts*, ~~UTAH L. REV. 2:327-356, at p. 330~~, 2 UTAH L. REV. 327, 330 (2010).

⁷³³ See Hand, *supra* note 381, at 56 (calling for "a board of experts or a single expert, not called by either side," to "advise the jury of the general propositions applicable to the case").

⁷³⁴ See Findley et al., *supra* note 3, at 306.

has also been presented, in contrast, is the lack of evidence-based literature for alternative hypotheses—such as hypoxia, the immature dural vascular plexus theory, dysphagic choking, and “neck” injury.

AHT/SBS cases are complex, difficult cases. Physician’s engaged in such cases do “acknowledge the complexities” and “consider alternative medical causes.” It is exactly those precepts that delayed the medical and societal recognition of abusive injury as the correct diagnosis in the early historical cases. Now some scholars argue for a return to those times as a “path forward.” But diagnostic and testimonial abstinence has had its day on the scientific stage. It has had its day on the differential...and science has ruled it out.